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Vol. II DECEMBER, 1926 No. 2

THE AMERICAN HEART JOURNAL

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PUBLISHED BI-MONTHLY

UNDER THE EDITORIAL DIRECTION OF

THE AMERICAN HEART ASSOCIATION

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PUBLISHED BY THE C. V. MOSBY COMPANY, 3523-25 PINE BLVD., ST. LOUIS, U. S. A

Entered at the Post Office at St. Louis, Mo., as Second Class Matter.

[NUMBER TWO OF A SERIES]

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The American Heart Journal

VOL. II DECEMBER, 1926 NO. 2

Original Communications

A CLINICAL STUDY OF CONGENITAL HEART DISEASE IN CHILDHOOD*

EDITH I. M. IRVINE-JONES, M.B., CH.B. (EDIN.)
SAN FRANCISCO, CALIF.

SINCE the middle of the eighteenth century, there has been an increasing literature on the subject of congenital heart disease. Studies of this condition made by such brilliant observers as Morgagni, Meckel, Rokitansky, Keith, and Abbott have almost invariably been made in the autopsy room, and little attention has been paid to the symptomatology during life. It is obvious that a knowledge of the pathology of any particular case is essential to the understanding of the mechanism underlying the symptoms. Few have studied the reactions of the congenital cardiac to the normal strain of life or to the extra strain of disease, and relatively little is understood of the significance of the occurrence of cardiac anomalies in a family.

With this in mind, an entirely clinical study is justifiable since it is true that we are yet incapable of arriving at an accurate antemortem diagnosis, and since our diagnosis and prognosis depend less on the anatomical lesions than on the clinical reaction of the patient to fatigue and intercurrent disease.

This study is based on a hundred cases of congenital heart disease seen during the last twelve years in cardiac clinics of the University of California Hospital, the Children's Hospital of San Francisco, and the Telegraph Hill Dispensary of San Francisco. Out of a hundred cases examined during the last ten years, forty-five are known to be alive, twenty-nine have died, and the fate of the remaining twenty-six is unknown.

Congenital heart disease has little predilection for any particular sex, though boys were affected in 56 per cent of the cases studied and girls in 44 per cent.

*From the Department of Pediatrics, University of California Hospital, San Francisco, California.

In a relatively large proportion of the children, the diagnosis was made within the first year of life, and many times in the first six months. The early diagnosis was not limited to patients suffering from the graver forms of congenital heart disease. The blue dyspneic baby may live to adult life, while an apparently normal child whose cardiac anomaly is an accidental finding may suddenly die during a mild upper-respiratory infection.

While the mortality is striking, the morbidity is even more so. We find that 71 per cent of the patients are disabled in various ways. In the majority of instances the crippling is obviously of cardiac origin; in eleven cases mental defect was the predominating factor, while severe nutritional and other disabilities were present in the remaining children. A survey of congenital cardiacs of all ages shows a striking nutritional defect. The newborn is almost the only exception, and one is struck with the very normal weight of such children at birth, the average being 6.73 pounds. As age increases, the nutrition in many cases fails, and we find fifty-six of the children over three months showing an average of 26 per cent underweight, twelve of the cases being over 30 per cent underweight. Only five of the patients were overweight, and glandular dystrophy accounted for this in two of these. The weight disturbance is not the only sign of a defective nutrition; the whole appearance of the child is suggestive. Pinched, old features and miserable posture are present in many cases, while definite dwarfing occurs in 34 per cent of the series.

During the first two years, one finds several departures from normal development. About one-third of the babies are slow, and this group can be divided into two sections: the children who are uniformly slow and who in many cases are defective, and the children who teethe and talk early but cannot sit up or walk until long after the usual time. These last are typical of the cases showing cardiac disability early in life. The later development of the child is not infrequently retarded, though our adolescent children show no marked infantilism, the menarche is not delayed, and development seems to progress normally. By puberty, of course, most of the severely defective children are dead and the survivors are those most nearly approaching compensation.

SYMPTOMS

In evaluating the symptoms of congenital heart disease in early infancy, two features stand out: (1) the cyanosis, which occurred in thirty-one of the infants, and (2) the nutritional difficulties, which were prominent in twenty-seven of the cases. That the feeding difficulties were dependent on the heart condition is made almost certain by their uniform occurrence and by the fact that almost three-quarters of the babies were breast-fed for at least three months and

so had a fair chance to develop, so far as feeding was concerned. Other symptoms which were prominent at birth included dyspnea, asphyxial convulsions, excessive crying. Hyperesthesia of the precordium was an interesting feature in one child; touching the skin initiated a convulsion. Two of the babies could sleep only on their left side, while placing them on their back elicited extreme discomfort.

In later life, cyanosis is perhaps the most common symptom associated with congenital heart disease. About 70 per cent of our children showed this symptom in varying degree, from the intense plum color present in four of the cases to the bluish pallor of many of the newborns and the characteristic high color of several of the older children, where floridity changed to cyanosis on exertion. As to the time of origin of the cyanosis, thirty-one of the sixty-nine cyanotic children, or 45 per cent, were "blue babies," the cyanosis being present at birth or developing within the first three days. Eleven more became cyanotic during the first year, and a further eleven did not show the symptom until later. In almost all of these cases cyanosis was a gradual development. Sudden permanent cyanosis occurred in three cases, once after a pulmonary infection where presumably an arteriovenous shunt was reversed by the increased pulmonary pressure. In two other cases sudden constant cyanosis followed surgical operation. A striking feature in five of the cases was the intense terminal cyanosis, where pallor had been an invariable feature during life. Many have looked upon the degree of cyanosis as an indication of the gravity of the prognosis. That the relation at least is not constant is shown by the figures (Table I) on the mortality among these children with varying degrees of cyanosis.*

TABLE I

CYANOSIS	NO. OF CASES	NO. DEAD	PER CENT DEAD
Not cyanosed	31	6	19.3
Floridity and transient slight cyanosis	7	0	
Slight cyanosis, constant	28	12	42.8
Moderate cyanosis	12	4	33.3
Marked cyanosis	18	6	33.3
Extreme cyanosis	4	1	25.0

It will, therefore, be seen that the highest mortality occurred among the slightly cyanosed children, while those extremely blue showed rather less tendency to die. The cyanosis when present is seen most strikingly on the malar prominences and the ears in the older children, on the entire skin of babies and of the older cases with the more extreme grades of cyanosis. The mucous membranes and conjunctival surfaces are frequently involved, the gums are spongy and the eyes

*These figures do not include terminal cyanosis within a few hours of death, as of course such cases fall into the noncyanosed group during life.

suffused. Numerous venules stand out with great distinctness in the palate and pharynx and the skin surfaces generally.

Vaquez[1] makes a statement that although cyanosis generally causes arrested development, it is not necessarily incompatible with perfect mental and physical growth. We have instances of both these sequels, proof of the latter being one or two youngsters whose blueness was so marked that they were refused employment, yet whose physique and alertness were normal. Arrest of development is, however, the more common finding. Bard and Curtillet[2] (quoted by Vaquez) mention the agonal reopening of a patent foramen ovale as the cause of the terminal cyanosis. This ingenious explanation does not invariably hold. One of our cases clinically showed all the signs of a congenital heart during life and had the accepted physical signs of a patent foramen ovale (though this diagnosis was not made antemortem). During the last thirty-six hours of life, cyanosis was intense and autopsy showed a widely open foramen ovale as the only lesion. In this event the foramen must have been open from birth, the blood shift must have been of the gamma-alpha type (from left to right) or perhaps from right to left, yet involving less than the classical third described by Lunsgaard and Van Slyke as being the minimum shift in the production of cyanosis. A terminal pneumonia in this case aggravated or initiated the alpha-gamma shunt.

We have never seen the type of cyanosis described by Variot[3] in its classical form with a pathological basis similar to that which he described. Of the cases which showed asphyxial attacks with intense cyanosis and syncope, none were entirely free from cyanosis or floridity in the intervals. Retinal cyanosis is simply a manifestation of cyanosis elsewhere. In the thirteen instances where the eyegrounds were examined, all the subjects showed some degree of skin cyanosis and ten showed definite retinal changes. The changes consisted mainly of a darkened color of the retina and dilated tortuous vessels, but without hemorrhages. One case in addition showed a neuroretinitis. Two of the cyanotic children examined showed no obvious retinal cyanosis. This finding does not substantiate the theory that retinal cyanosis is an important precursor of skin cyanosis. Nagel[4] has suggested eyeground examination as an ingenious method of differentiating between pulmonic stenosis and defects entailing admixture of blood, his theory being that in pulmonic stenosis the retinal arteries will be normal and the veins congested, while in septal defects both vessels will show changes.

Twelve autopsies are included in this series, and in most there was adequate reason for the cyanosis in the multiple defects combining stenosis and free admixture of blood. Clinically, it is futile to attempt to weigh the different factors present in the mechanism of this classical symptom. We are interested in the theory advanced by

Meckel that cyanosis occurs only in the presence of admixture of blood and that if it is absent no admixture is occurring. Professor Vaquez, who supports Meckel's theory, explains the lack of cyanosis in those children where there is, for instance, a septal defect, by the formation of muscular channels guiding the blood stream toward the normal orifices. The case we would like to mention in this connection is that of a fifteen months old baby who had no disability excepting malnutrition until two months prior to his death. There was no polycythemia and no cyanosis except as a terminal phenomenon. Cardiac insufficiency was shown by the occurrence of anasarca on three occasions, and slight albuminuria was present. Death was due to sudden cardiac failure. The autopsy showed a greatly hypertrophied two-chambered heart. It is difficult to imagine that in this instance no admixture of venous and arterial blood had occurred.

Holman,[5] without being so certain that admixture of blood is the only factor in the production of cyanosis, supports this theory, which is also the principle advocated by Maude Abbott. The part that pulmonary lesions play in the production of cyanosis is still uncertain, though dilatation of the lung capillaries with circulatory stasis has been noted in a cyanosed case of pulmonary atresia by George Carpenter.[6]

The cyanosis in the more prolonged cases is very probably due to a combination of factors, as we shall see when we consider the pulmonary complications of this condition.

In the case just quoted, the anasarca is interesting, as edema is one of the rarer findings in congenital heart disease and occurred in only two of our cases. Loutaud[7] states that in congenital heart disease the veins are hypertrophied and that edema does not, therefore, occur; this does not invariably hold, as we have seen.

The most prominent symptom, apart from the cyanosis, is the dyspnea which we observed in forty-seven of the children. The dyspnea generally occurs some time after birth and is definitely related in many instances to the early efforts at walking and later to the activities of school life. Seven of the babies were definitely dyspneic at birth, and additional feeding difficulties were therefore encountered.

Many authors have stressed the mental aspects of congenital heart disease, and we feel that mental deficiency adds greatly to the morbidity of the disease. Precocity has been given too great a prominence; we have seen only one child with congenital heart disease who could have been called in any way precocious. Psychical changes are frequent. The children are unlike their brothers and sisters, are harder to manage in the home, and definite mental impairment is present in a fairly large proportion. Twenty-nine of our patients showed a subnormal mentality and this figure we feel is modest, as

a considerable number of the children were very young and actual data as to their mental status could not always be obtained. The children belonging to this group fall into two classes: First, there are those whose backwardness is simply due to their impaired cerebral circulation—cyanosis, polycythemia and clubbing being generally present, and the mind is not imperfect but merely torpid. These children are difficult to interest and their attention is hard to keep. If given some problem to solve, they will do it very slowly but probably quite accurately. The intelligence quotient in those cases is around 80 per cent, but much can be done for them by individual tuition. The second group includes those where cerebral defect is present, where other congenital stigmata such as cleft palate, facial asymmetry and similar deformities are prominent. In this group the defect obviously depends on a defective anlage of the entire organism early in fetal life, when multiple defects, cardiac, skeletal and cerebral are caused by some inhibiting factor. The backwardness varies from a slight degree of mental retardation to idiocy. Mongolian imbeciles constituted seven of our series, and two other children were definitely mongoloid. The association of congenital heart disease and mongolism has been stressed by Garrod[8] and Von Hofe,[9] who found congenital heart disease in about 10 per cent of their Mongolian patients. Morley Fletcher and Parkes Weber[10] mention the other types of congenital mental defect which are associated with an abnormally high proportion of congenital cardiac patients and it is into this category that most of our cases fall. Most of the mentally defective children have multiple physical defects, and some look more defective than a mental examination rates them.

Pallor is a symptom not commonly associated with morbus cordis, yet it was striking in about a quarter of the cases. To what is this pallor due? In some cases the nutritional defect of the child afforded a reason for the pallor, while in some exercise and fresh air were insufficient on account of the physical disability. Definite anemia was present in some cases and it is interesting that as the children improved under a hygienic regime, cyanosis became more apparent. This is in accordance with the experimental work of Lunsgaard and Van Slyke[11] who state that in the presence of a severe anemia, cyanosis cannot develop. Pallor may be due to deficient arterial circulation such as occurs in aortic stenosis and in coaretation of the aorta. Here it is associated with other signs of deficient peripheral circulation, a small or absent pulse, impalpable aorta, cold extremities and signs of general weakness. Clubbing of the extremities is a symptom which appears in about one-fifth of the cases. It is invariably accompanied by cyanosis, usually of marked degree. The average age at which clubbing was first observed in our cases was five and one-half years. It is not therefore a symptom of congenital heart disease in

the earliest days. The youngest case we have in our records was a cyanosed baby of four months reported to show clubbing. We have seen two marked cases in children of nine and eleven months respectively. The degree of clubbing varies greatly, although in a majority of cases it was definite but not striking. Late clubbing was a feature of the case reported by Henry Christian.[12] His patient was a girl of twenty-six years, with multiple cardiac anomalies, who had no symptoms until ten years of age and no clubbing until twenty-four years. We have found in the literature no instances of neonatal clubbing. This is, of course, what one would expect. One of the earliest instances reported is a case of Wohl,[13] where clubbing was present at three and a half months in a baby with cor triloculare and transposition of the vessels.

Syncope is a fairly frequent symptom of congenital heart disease, and was present in fourteen of the cases. Most of the children lost consciousness and a few complained merely of sudden dizziness. No cyanosis was associated usually with the cases which showed vertigo, but of nine children who had definite syncope, eight were cyanosed and all eight suffered from frequent convulsions, obviously of asphyxial origin. In such cases after some slight strain the child feels dizzy and faint, turns blue, twitches, loses consciousness and on recovery is somnolent, limp and gray. Convulsions due to other causes are of course not included.

Angina is a symptom not generally considered in the heart disease of childhood, and especially of early infancy. In fourteen cases it was noted as a striking symptom. The majority complained of actual precordial pain, while in a few of the cases which occurred in infants the symptom was inferred from the continual crying and moaning of the baby. Hyperesthesia of the precordium is an interesting finding in one of the children, who screamed and became convulsed when the precordium was touched. Palpitation occurred in most of the cases who complained at some time of pain. This symptom differs in no respect from the same symptom in adults.

PHYSICAL SIGNS

Of the physical findings in congenital heart disease, the most constant and the most misleading and one of the least useful from any standpoint is the murmur which is present in 90 per cent of the cases. The ten cases where murmurs were absent included a case of dextrocardia with heterotaxy, where presumably the malposition was the only anomaly. The absence of the murmur was particularly a feature of the infants, and excepting the case mentioned above, all the patients were under one year. Autopsy diagnoses in these cases included wide patency of the ductus arteriosus, transposition of the great vessels, and idiopathic congenital hypertrophy, in both of which

last conditions murmurs are rare. It is interesting to remember that Owen and Kingsbury[14] believe that the murmur of a patent ductus is never present at birth, that the bruits of true congenital heart disease rarely appear before the end of the second week, and that those heard over the precordium at birth are functional in nature. Wusthoff,[15] on the other hand, notes that functional bruits are very rare under the fourth year of life, and he incidentally quotes a case of patent ductus arteriosus (with, however, a complicating open foramen ovale) where the murmur appeared on the third day of life. In most of the cases with murmurs, the bruit was systolic in time; in only one instance was a murmur limited to diastole present, while in sixteen the bruit was double or continuous. Stethophonic records will probably show a greater incidence of a continuous or double murmur than can be picked out by auscultation, but as we mentioned before, the occurrence and type of murmur is of little assistance clinically in determining the lesion deciding the prognosis. One of the factors which complicate greatly any effort at arriving at a diagnosis of the lesions of congenital heart disease is our complete ignorance of the degree of myocardial defect in those cases. When one is dealing with the cardiac changes which occur after rheumatic carditis, one is dealing with known factors. The heart, if the myocardium is not too severely injured, will compensate for the circulatory defect by dilatation and hypertrophy, and permanent enlargement occurs in many cases. No such reaction can be confidently anticipated in congenital heart disease; otherwise, many more congenital cardiacs would be symptomless. This failure of response or aberrant response of the congenital heart seems to show itself in four ways:

1. By the characteristic symptoms of the disease.
2. By the size of the heart.
3. By the abnormal electrogram.
4. By the varying exercise reaction of these children.

Cardiac enlargement occurred in sixty-four of our cases, but in only eighteen was it at all marked. The remainder were normal in size except for a few which were abnormally small, particularly in the older children. It is interesting that the children with hearts of apparently normal size were not the symptomless cases. This failure of response is partly due, in some cases at least, to the defective coronary circulation, as in several instances the coronary arteries arose from the venous side of the heart. In other cases one must postulate a congenital aplastic condition of the myocardium. It is this variability of the reaction of the congenital heart which makes fluoroscopic or roentgen-ray studies so difficult of interpretation. The chamber which should be enlarged if the diagnosis is correct may be actually smaller than usual. The pulmonary arch which you may ex-

pect to be small may be widely dilated. The electrocardiogram is in most cases abnormal, frequently showing an altered type of conduction and very often an altered response with either a diminished or increased excursion of the waves. One feels that the electrocardiogram when more fully studied will be of more value in diagnosis than any other individual factor. In 97 per cent of cases in which electrocardiographic studies were made, abnormal curves were obtained. The cases where a normal electrocardiogram was obtained in the presence of indubitable congenital heart disease are interesting in that the lesion was clinically a patent ductus arteriosus in two cases where presumably a normal electrocardiograph reaction might be obtained. The third case, which was followed from birth, exhibited in its earlier years all the signs and stigmata of congenital heart disease; at present at six years, excepting for the nutritional changes, multiple other anomalies, and a faint bruit after exercise, no symptoms of congenital heart disease remain.

The alterations in the electrocardiogram may be summarized thus:

1. Splitting of the P-wave—12 per cent.
2. Right axis deviation—75 per cent.
   In children under one year this was only considered abnormal if the White index was considerably over 12.
3. Left axis deviation of abnormal degree—20 per cent.
4. Abnormally large QRS complexes—45 per cent.
   Accurate calibration of the machine is an essential in gauging the degree of this change.
5. QRS variations—slurring—16 per cent. QRS variations—definite interventricular block—21 per cent.
6. T-wave slurred—4 per cent.
7. Atrioventricular block—3 per cent.
   (NOTE.—The classical mirror image picture of dextrocardia with heterotaxy was present in the only example of this condition where an electrocardiogram was possible.)

Lewis[16] has stressed the association of greater amplitude of the waves in congenital heart disease and for a considerable time interest has been taken in the right preponderance which was first noted by Steriopulo[17] and is fully discussed in a paper by Hugh McCulloch.[18] Abnormalities of the P-wave are thought by Alexander, Knight and White[19] to be very suggestive of pulmonic stenosis.

The statistics which we have quoted bear out the belief that the cardiac muscle is extensively affected in many cases of congenital heart disease and that the response of the heart cannot be anticipated

in the sanguine way with which one studies heart disease due to other causes. The third interesting factor in one's consideration of the cardiac muscle is the exercise reaction of the child with congenital heart disease. A normal individual reacts to fairly strenuous exercise by flushing, dyspnea, tachycardia, and raised systolic output.

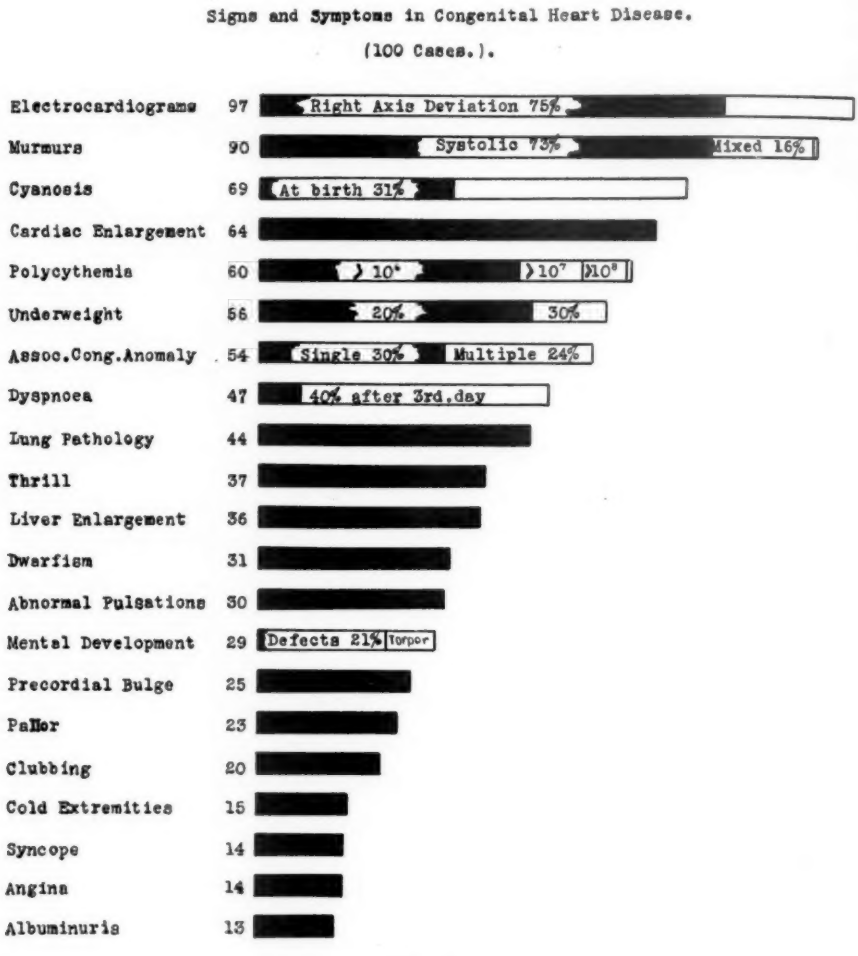

Fig. 1.

Normal conditions are regained after two to two and a half minutes. The rheumatic heart, depending on the myocardial impairment present, shows a corresponding delay in the return of the preexercise condition and in some cases the blood pressure continues to rise a little after exercise has ceased. In congenital heart disease we find a variety of responses, and this is what one expects from the variety of lesions present. About 25 per cent of the children tested showed

little difference from the normal. The other 75 per cent showed a different reaction. Dyspnea and cyanosis are the most prominent symptoms resulting from exercise, while tachycardia and delayed return of the blood pressure to the normal are present in about half of the cases. A few show a sudden drop to zero in the diastolic pressure, and this has been noted in several instances to be unilateral. A curious reaction which requires further study is the fall of the systolic pressure which occurs in some cases after exercise. These children, though cyanotic, dyspneic and on the verge of syncope, show a blood pressure similar to that obtained before exercise or even five to six points lower. The symptoms and signs in some of these are suggestive of interventricular septal defect so that one possible explanation of this unexpected reaction would be the increased action of the left ventricle sending blood through a septal defect to a greater degree than before, thus leaving less blood for the systemic circulation.

OTHER PHYSICAL ANOMALIES

An important clinical finding in congenital heart disease, which has until the present received rather scant attention, is the frequent presence of deformities other than cardiac. Over half of the children showed various congenital defects, and in twenty-four of the cases the anomalies were multiple.

A rough idea of the type of defects found may be gauged from Table II.

TABLE II

Total showing defects	54 cases
Showing multiple defects	24 "
Defects of the extremities	9 "
(Club foot, congenital dislocation of the hips, syndactyly, etc.)	
Defects of the head	8 "
(Premature synostosis, marked asymmetry, macrocephaly, etc.)	
Mongolism and its characteristic defects	7 "
Rib anomalies	6 "
(Fused ribs, absent ribs, supernumerary ribs, and in 3 separate cases the 3rd left rib was bifid)	
Palate (cleft or very highly arched)	6 "
Testicles undescended	5 "
Ear anomalies	5 "
Spinal anomalies	4 "
Dental anomalies	4 "
(Suppression of incisors, etc.)	
Congenital tumors and cysts	3 "
Nystagmus	3 "
(Unassociated with mental defect in 2 cases; mental defect in the 3rd)	
Thymic enlargement	3 "
Visceral anomalies	3 "
(Liver, spleen, lungs)	

Other anomalies appearing less frequently are those of phimosis, congenital herniae, labyrinthine aplasia and nasal septal defects.

Attempt has been made to analyze these defects and to correlate them with the cardiac deformities to determine whether both occurred at the same time in fetal life. If such could be shown, one would then seek some factor external or internal which operated at this time producing the multiple defects. For example, one of our cases which showed postmortem a patent interventricular septum as the only cardiac defect had also a marked degree of club foot. Now the interventricular septum is formed about the fourth week of fetal life, at which time the lower limbs are not yet differentiated. To cite another example: A child with congenital heart disease and a cleft palate showed postmortem a big defect of the interauricular septum. The interauricular septum is partly present at the fourth week of fetal life, showing therefore that the error of development occurred before that time, yet palatal development does not occur before the tenth week. One could multiply such instances indefinitely. One may deduce from these instances that the arresting factor operates at no one point of fetal life, but has a constant inhibiting effect on the embryo.

A common accompaniment of congenital heart disease and the origin of which is still obscure is the polycythemia, which is often striking. Our figures are based on fifty-seven cases, as blood counts were done only on that number. Thirty-three children, or fifty-nine per cent, showed polycythemia of five and a half million or over, our highest count being 10,688,000, with 136 per cent hemoglobin. The red count varies slightly, depending on whether the blood is taken from capillaries or by venipuncture. The hemoglobin, although generally raised in polycythemia, has no constant ratio to the red cells and in some cases is very markedly low. Polycythemia is not classically a symptom of infancy. We had one case, however, a child of three months, who showed a red count of eight million and who died of cardiac failure. As to the causes of polycythemia, two of the theories at least require consideration. First, is the polycythemia due to the concentration resulting from circulatory stasis? Bie and Marr[20] have shown that hydremia exists in congenital heart disease and we have found a constantly low white count, both of which facts are against the theory of concentration. Secondly, is the polyglobulia a compensatory reaction to the cyanosis? If it were so, cyanosed children would show some degree of polycythemia, but this does not hold. Sixteen of our cyanosed children, or 27 per cent, showed no polycythemia whatever. Most of the children with polycythemia were cyanosed, however, although we have four instances where polycythemia was present and cyanosis absent. In these last four cases, a possible explanation may be that the polyglobulia exactly balanced the oxygen deficiency so that cyanosis did not occur. We believe, therefore, that polycythemia is probably compensatory to the oxygen deficiency, but that the compensation does not necessarily occur. The

place of polycythemia in the prognosis is interesting in that our statistics would indicate no special tendency for this symptom to be associated with a poor prognosis, as is so frequently stated. It is probably a natural phenomenon resembling cardiac or kidney hypertrophy which compensates for some related deficiency. The child whom we quoted as having a red count of ten and a half million is disabled mentally and physically but has reached the age of thirteen and is able to attend school all day.

Trophic disturbances are recognized as occurring in congenital heart disease to a large extent. They include the nutritional changes, the infantilism or the premature senility, the clubbing of the fingers and toes, the changes in the nails, and the cerebral torpor. Pigmentation of the skin from prolonged cyanosis is present in a few cases, and changes in the texture of the hair may also be noted.

The classical signs include, of course, the murmur which we have previously discussed. A precordial thrill was present in 37 per cent of the cases. This is a feature which is not commonly noted in the youngest patients. Abnormal pulsations whether cardiac or extracardiac were present in about one-third of the cases, and precordial bulge in about the same number. The characteristic deformity is usually left sided, although we have several cases where the maximal bulge was on the right side of the sternum and rare cases where it was central in position.

Lung pathology is a striking concomitant of congenital heart disease and it is not frequently stressed that it forms the most important factor in the prognosis of the disease. Forty-four per cent of this series showed lung changes (Table III).

TABLE III

Bronchopneumonia present at the time of examination	8 cases
Collapse and atelectasis	8 "
Fibrous thickening	7 "
Bronchial gland enlargement	7 "
Tuberculosis	7 "
Persistent râles without other findings	7 "

No comment is necessary except perhaps that the incidence of tuberculous lung infection is less prominent than older statistics show.

PROGNOSIS

The prognosis in congenital heart disease is one of the most difficult problems which the pediatrician has to face. It is impossible to give parents any but a rather grave outlook. Maude Abbott's estimate of the life limits of a child with pulmonic stenosis is fifteen years. Cases with patent ductus arteriosus, where symptoms are commonly absent and the prognosis is generally considered fair, are still liable

to ulcerative endocarditis. Sudden death occurs not infrequently in the so-called "mild" cases of congenital heart disease.

That complete recovery from a congenital lesion is possible seems dubious. We have quoted one case of clinical recovery and John Thomson[21] in 1901 published a case of probable patent ductus where clinical signs entirely disappeared and where closure of the ductus was thought probable. Until the mechanism in the production of murmurs is better understood, until we know the reason why a patent ductus in antenatal life produces no bruit while later the murmur is classical, we cannot depend on clinical signs alone for such deductions.

Our own statistics show that twenty-nine of the hundred cases are known to have succumbed. From these cases and from a study of the surviving children, one can draw various conclusions regarding the prognosis:

1. *The Age at Death.*—Half the deaths occurred under six months of age. The other fatal cases occurred between six months and twenty-one years, with relatively little age preponderance. One may therefore conclude that a baby surviving the first six months has doubled its chances of life.

2. *The Cause of Death.*—Cardiac failure as such is not the most common cause of death, although it accounted for nine of the fatal cases. There is no special age when cardiac failure is particularly common. The largest number are found naturally when the death rate is highest, but deaths from heart failure without apparent infection are noted at nine years and twenty-one years. Pulmonary conditions accounted for thirteen of the deaths, although naturally a cardiac element is present in many of these. The age incidence here lies between two weeks and ten years. Pneumonia, bronchitis, and tuberculosis were the lesions in most of the cases, but tuberculosis caused death in only two cases. Endocarditis caused death in two cases and was contributory in one instance. Infections of other types and hereditary lues caused death in the remaining cases. Of four cases of congenital lues associated with congenital heart lesions, three are dead. These facts point their own moral. Heart failure takes a big toll of the children, but infections which are to a great extent preventable are the greater danger.

Pertussis is a disease which is responsible for a great deal of disability in congenital heart disease. It does not kill the patient, but a striking number of children develop their first dyspnea and cyanosis after whooping cough and nutritional difficulties are also exaggerated. It is therefore a disease to be guarded against and treated energetically when it occurs.

3. *Albuminuria* is a factor which is associated strangely enough with a rapidly fatal outcome, and here the connection is not very clear.

Thirteen of the entire series showed albuminuria in the routine urinalysis, and the amount of albumin in the great majority of cases was slight. It is significant that eleven of those cases subsequently died. Although it is generally thought that polycythemic children are apt to show orthostatic albuminuria, this is not usually true, as only four of these children had a faint trace of albumin. Casts were rarely found.

4. The fourth factor which we wish to discuss is the place which anesthesia or surgery has in affecting the prognosis. Thirty per cent of our series had anesthetics, mostly for tonsillectomy, and the anesthetic in most cases was ether. In no cases did permanent ill effects follow, although one child who was tonsillectomized in the country had a slight syncopal attack during the operation, and cyanosis was present in one of our cases during a cleft palate operation. In the majority, the color and pulse were unusually good and the bleeding was no more prominent than in normal children. One of the patients who clinically showed a patent ductus and who stood a tonsillectomy without untoward effects has become decompensated slightly after a cesarean section followed shortly by a laparotomy. It will be obvious from this that anesthesia and operation are of no more risk to a child with congenital heart disease than to the normal healthy youngster.

5. *Cyanosis and polycythemia,* although associated in many minds with a grave outlook, are, as we have seen, of little or no importance in the prognosis.

The morbidity of congenital heart disease is as serious a consideration as the mortality. One will recall that seventy-one of the patients were definitely disabled. Only six showed no disability, while in fifteen of the infants no estimate could be made. In most of the cases the crippling was cardiac in origin, although eleven of the cases showed relatively less physical disability but considerable mental retardation.

In considering the prognosis one must gauge the danger to life. One must consider the age of the patient and the presence or liability to infections of any type, although those of syphilis, ulcerative endocarditis and pulmonary disease are the most deadly. One must especially watch for the presence of albuminuria. Not less important in the prognosis is an estimate of the physical and mental handicap of the child that survives.

ETIOLOGY

I would like for a moment to take up the question of the etiology of the condition as we know it. Maude Abbott[22] divides her cases of congenital heart disease into two big groups, those due to arrested growth, and those resulting from fetal endocarditis. Keith,[23] on the other hand, takes the stand that probably all the lesions are due to developmental

arrest. On reviewing this present series, we see that 54 per cent of the children had other congenital anomalies elsewhere in the body, and this would incline one to the view of some common inhibitory factor. That it is not a developmental accident occurring at some point in fetal life I have shown by the time differences in the origin of the deformities. It must therefore be due to some baneful influence acting throughout fetal life,—maternal disease, uterine exhaustion from any cause, or hereditary stigmata. When one considers the question of maternal disease, one finds that 13 per cent of the mothers were actually ill during pregnancy and four further cases were suffering from luetic infection. Actual uterine disease is known of in only two cases. Chronic heart disease was present in eight of the parents. Hereditary stigmata were mentioned in only three cases and included congenital dislocation of the hips, congenital elevated scapula with scoliosis and syndactyly. The popular idea that the congenital cardiac is often the last child of a long series of pregnancies is a mistaken one. The old age of the parents as a factor in the production of the disease is also a fallacy. The average age of the mother was twenty-eight years, of the father thirty-one years, and the average place in the family was second or third. In thirty-three cases the congenital cardiac was the first child. It is interesting to note, however, that in only six families was a normal child born after a congenital cardiac, so that the uterus if not exhausted by multiple pregnancies or senility was probably exhausted by some other factor. A point supporting this statement is the striking amount of ill health in the brothers and sisters of congenital cardiacs. In four instances two congenital cardiacs occurred in one family, deformity of the extremities was fairly common, and rheumatic heart disease occurred in six siblings. We would like to note, however, that our series included three pairs of twins; in every case the normal twin showed absolutely no trace of disease.

We are inclined to support the views of Keith as to the developmental nature of the lesions, although until more definite correlation can be obtained the prophylaxis will be uncertain. At present it would seem to consist of a hygienic regime, particularly in the earliest weeks of pregnancy, the prevention of acute infection, and proper care of chronic disease in the mother.

MANAGEMENT

With the solution of the etiology, the prophylaxis will also be solved. Until this time, it remains to palliate the mental and physical crippling which is so frequently present. Feeding difficulties of varying severity were present in a third of the children. Babies become dyspneic after nursing only a short time. Food was refused and vomiting was present in almost all the cases. The feeding schedule in these children should obviously be arranged to give them small amounts of a high

caloric food at rather more frequent intervals and the difficulties of actual nursing should be minimized. Upper respiratory infections should be guarded against and the tonsils if diseased should be removed. Children with congenital heart disease should frequent as little as possible crowded places where infections, such as pertussis and colds are rife.

In preventing infections—colds, fatigue and exposure, as predisposing factors, should be avoided; and the gradual supervised hardening of the child is a most important measure. Sun baths should be instituted, and physiotherapy given at home and regularly checked in the clinic is of value. No exercise is advisable until a child is gaining weight and feeling well. Digitalis has been suggested by Morse[24] as of value, but we cannot see any possible reason for its administration, first because one cannot foresee the effect of digitalis on a heart in which the condition is probably normal, and second because unless one is familiar with the circulation in the particular heart in question any response which the myocardium may show to the stimulus may force the circulation through some of the anomalous channels and decrease the systemic circulation with fatal results. Oxygen by the usual method is useless in the cyanosis of congenital heart disease. This has been pointed out by Maude Abbott,[25] who suggests the use of intravenous oxygen as a temporary measure. The education of these children is the biggest problem. If they are almost symptomless no special arrangements need be made, although it is generally advisable to avoid competitive exercise. Where great disability is present, special teaching should be instituted so that the children will get the maximum information with the minimum waste of energy.

CONCLUSIONS

In conclusion, we would emphasize some of the outstanding features of our analysis: The large number of cases showing other anomalies lead us to believe that the cause of congenital heart disease lies in a defect of the germ cell or chronic disease of the maternal tissues rather than in an inflammatory condition of the fetal endocardium. Abnormalities in the electrocardiogram constitute the most prominent and constant clinical finding. The older idea that the prognosis varies with the cyanosis and polycythemia is inaccurate. Frequent respiratory infections and albuminuria do however give a poor prognosis. Half the deaths occur under six months of age and the prognosis rests partly on this factor. Tonsillectomy should be done in all cases where the tonsils are diseased and where infections are frequent. Anesthesia and operation are as well borne by congenital cardiac patients as by normal children.

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PAROXYSMAL VENTRICULAR TACHYCARDIA

Report of an Unusual Case*

T. Duckett Jones, M.D., and Paul D. White, M.D.
Boston, Mass.

THE number of cases of paroxysmal ventricular tachycardia, though as yet small, has been substantially increased during the past several years because of the wide use of the electrocardiograph. The literature, while not voluminous, has been covered by numerous writers, notably Robinson and Hermann,[1] Marvin and White,[2] Wolferth and McMillan,[3] Major and Wahl,[4] and Gilchrist.[5] In order properly to understand the mechanism of such paroxysms it seems desirable to have on record as many recognized cases as possible with complete electrocardiographic studies, together with the individual peculiarities of the cases. Especially is it of value to ascertain the prognosis, and the treatment which is most efficacious.

The case herein reported is of considerable interest because of the variations from the usual forms of ventricular tachycardia, and the good response to therapy.

CLINICAL RECORD

History.—S. F., male, aged 18 years, single, student, was referred October 23, 1925 for cardiographic study by Dr. Paul Means, because of repeated paroxysms of tachycardia. The condition was found in a routine physical examination at school, and not because of any discomfort or distress of the patient. He has been quite healthy and active all of his life. Pneumonia in 1916 and again in 1920 have been his only serious illnesses. Tonsillectomy was performed four years ago. There is no history of rheumatic fever or chorea. At the age of five years his father, a physician, noted the cardiac condition, which, according to his mother, is somewhat better than formerly. Care as to exercise and attention to general health have been the only measures instituted. The cardiac condition was found again a year ago upon entering a preparatory school and again recently upon entering college, as noted above. His only symptom has been a consciousness of frequent, almost continuous, spells of very rapid heart action. No discomfort has resulted, and there have been no signs whatever of cardiac failure. He states that he has felt best while performing strenuous exercise, notably mountain climbing.

Physical Examination, October 23, 1925.—The patient is a somewhat slender, well-proportioned young man, who shows no evidence whatever of physical discomfort. Heart examination reveals the following: Apex impulse maximal in the 5th interspace 10.5 cm. to the left of the midsternal line. The left border of dulness corresponds to this. In the 3rd space the left border of dulness is 6 cm. from the mid-line. The mid-clavicular line is 8.5 cm. Teleoroentgenograms confirm this enlargement, and there is a cardiothoracic ratio of 55.9 per cent. In the upright position there are no murmurs, but a definite third sound is heard. Lying down there are no pathognomonic murmurs either before or after exercise. There is a very rapid rate—160 per minute—at both the apex and wrist, with a very definite irregularity. Blood pressure is 140 mm. mercury systolic and 80 mm. diastolic.

*From the Cardiac Clinic, Massachusetts General Hospital.

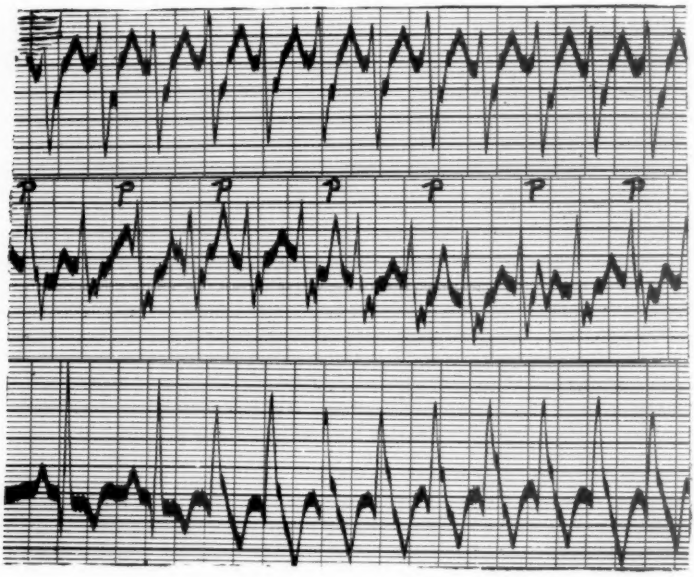

Fig. 1.—(Plate 10,359.) Leads I, II, and III. October 23, 1925. Paroxysmal ventricular tachycardia. Ventricular rate 160, auricular rate 95. P-waves are easily seen superimposed on ventricular complexes in Lead II. Note paroxysm of tachycardia in Lead III, preceded by two normal complexes. (In this and all succeeding electrocardiograms, distances between ordinates represent 0.2 second, distance between abscissae 10-4 volts.) Reduced to ⅔ size, as are also the remaining figures.

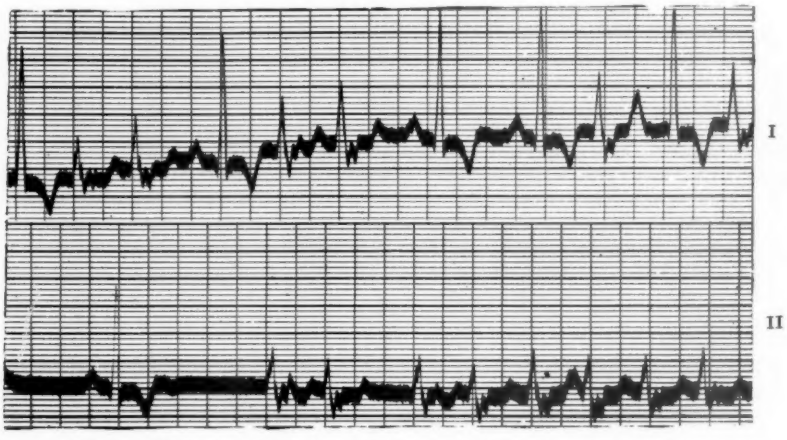

Fig. 2.—(Plate 10,362.) Lead II. October 23, 1925. Normal rhythm interrupted by ectopic ventricular beats, at times in short runs. Of the same general shape as seen in Fig 1. Taken immediately after exercise. The rate dropped from 160 to 130. Deeply inverted T-II.

Electrocardiographic tracings and the response to quinidine sulphate are outlined below, covering a period of some eight months:

October 23, 1925.—Ventricular paroxysmal tachycardia with a ventricular rate during the paroxysms of 160 per minute, and an auricular rate of 95. In Lead II the P-wave was easily seen superimposed on ventricular complexes at a rate of 95 (see Fig. 1). After exercise (see Fig. 2) normal rhythm occurred, interrupted by ventricular pre-

mature beats at times in short runs. The heart rate was definitely slowed and the P-wave varied from + 4.5 mm. to + 2 mm. as the auricular rate slowed. The T_2 was inverted and measured – 4 mm. Quinidine sulphate 0.6 gm. daily was prescribed.

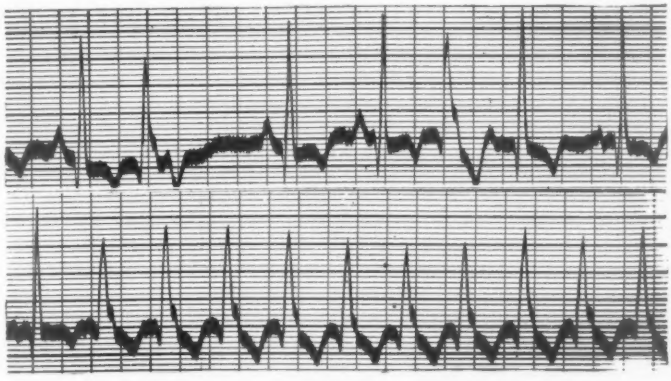

Fig. 3.—(Plate 10,363.) Lead III. October 23, 1925. Same as Fig. 2. Normal rhythm with frequent ectopic ventricular beats. Paroxysm of tachycardia begins in the bottom strip. P-wave changes greatly.

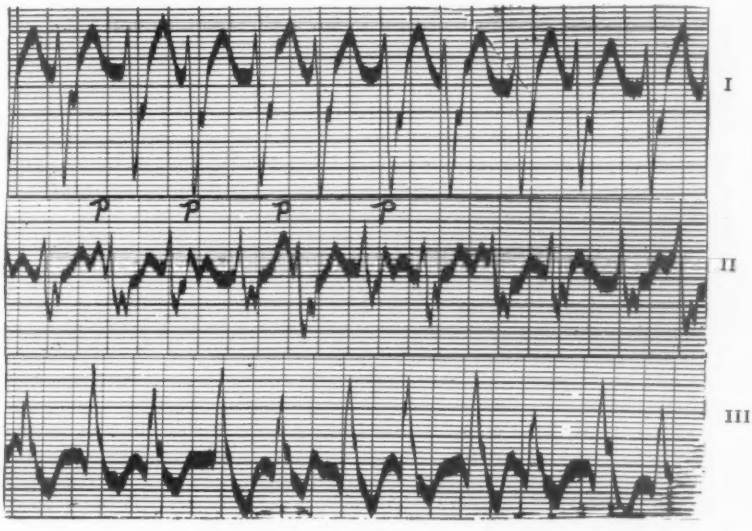

Fig. 4.—(Plate 10,421.) Leads I, II, and III. November 3, 1925. Paroxysm showing definite bigeminy, alternate beats arising from different ventricular foci (?). Bigeminy inconstant.

November 3, 1925.—Ventricular paroxysmal tachycardia, with a ventricular rate of 140, and auricular rate of 95, the P-waves being superimposed in Lead II as before (Fig. 4). Bigeminy was present, possibly due to a second focus in the ventricle. The interval between the 1st and 2nd beats was 0.4 second, and between the 2nd and 1st beats 0.47 second. After exercise three normal beats were seen with a high P_2 (+ 5 mm.), inverted T_2 (– 4 mm.) and a rate of 95. One

ectopic ventricular contraction preceded this rhythm, with an interval of 0.66 second at the end of a paroxysm. Bigeminy was not always constant. The patient reported that for one day he had a regular pulse with a rate of 70. Quinidine sulphate was increased to 1.2 gms. daily.

November 10, 1925.—Normal rhythm, rate 90. There was a high, wide P-wave throughout, and the T-wave was deeply inverted. Right axis deviation, with an index of – 20 and an angle of 90°, was found. No paroxysms of tachycardia and no premature beats occurred. The patient had been conscious of no rapid heart action or irregularity

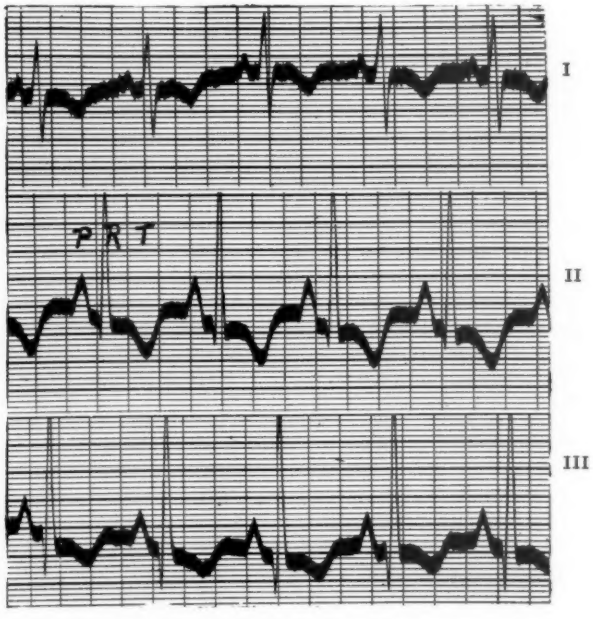

Fig. 5.—(Plate 10,531.) Leads I, II, and III. November 10, 1925. Normal rhythm. Rate 80. Note the high wide P, and deeply inverted T seen in all tracings of constant normal rhythm.* This record was taken after one week of quinidine sulphate 1.2 gm. daily.

during the week, and felt that his pulse had been constantly below 100. Quinidine sulphate was stopped.

November 17, 1925.—Ventricular paroxysmal tachycardia as when first seen. Patient conscious of irregular, rapid action during the entire week. Quinidine sulphate 1.2 gm. daily again started.

November 24, 1925.—Normal rhythm, rate 80. P- and T-waves as before. Two 0.2 gm. doses of quinidine were taken before his rate slowed down, and it remained down the entire week. Quinidine sulphate 1.2 gm. daily for one week, then 0.6 gm. daily, advised.

December 8, 1925.—Ventricular paroxysmal tachycardia as previously, with a ventricular rate of 145. The heart rate had remained

*The records showing constant normal rhythm are not unlike those seen in some congenital heart cases. This has been considered, but no clinical evidence of a congenital defect could be demonstrated.

regular and slow until the dose of quinidine was reduced, when the rate increased noticeably. Quinidine sulphate 1.2 gm. daily was advised for several months.

February 1, 1926.—Short runs of ventricular tachycardia, with a ventricular rate of 130, and auricular rate of 90. The patient reported that he had been quite well, that his rate never was faster than 130 and that it remained below 100 as a rule if he had his quinidine (1.2 gm.) regularly.

March 29, 1926.—No change since last visit. Short paroxysms with the ventricular rate not faster than 130. After exercise, normal rhythm with a ventricular rate of 100 was found. He had been in good health and felt that quinidine definitely kept his pulse rate down.

June 11, 1926.—Heart regular at 110. Felt well and had been very active. Had taken quinidine 1.2 gm. daily. Quinidine was omitted.

June 28, 1926.—Felt almost as well without quinidine as with it, but his rate was 136 when recumbent, and varied from 100 to 120 in the upright position, with definite irregularity. He was advised to take 0.6 gm. of quinidine daily and to report in several months.

SUMMARY

The case is recorded of a patient with paroxysmal ventricular tachycardia which presented some very unusual features. Frequent electrocardiographic studies disclosed interesting changes. Records are shown in which ectopic ventricular beats interrupt the normal rhythm, and are similar in form to the complexes during the paroxysms. The auricular waves are clearly outlined during the paroxysms, in Lead II especially, apparently arising from the sinoauricular node and at varying rates. The paroxysms are short and similar to runs of ectopic ventricular beats rather than to prolonged tachycardia. Quinidine sulphate, when given in sufficiently large doses (1.2 gm. daily), reduced the ventricular rate from 130 to 100 or less, restoring normal rhythm, while without the drug the rate was frequently 160 to 180. Daily doses of quinidine of 0.6 gm., while at times effective, did not maintain normal rhythm. There was no change in the patient's general condition while taking the drug except that he felt certain that his heart was beating much more slowly than usual.

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CLINICAL ASPECTS OF CONGENITAL ANOMALIES OF THE AORTA*

JOHN T. KING, JR., M.D.
BALTIMORE, MD.

THE congenital or developmental malformations of the thoracic aorta may be considered in three categories. First, there are occasional instances of widespread distortion of the cardiovascular system, in which an anomaly of the aorta plays a relatively insignificant rôle. Such cases are not common; they are not susceptible of accurate diagnosis during life, and I shall therefore not discuss them in this paper. A second group includes the cases of generalized hypoplasia of the aorta and its main trunks. The third type of malformation is the stenosis of the isthmus of the aorta, known usually in the English speaking literature as coarctation of the aorta. By the "isthmus" is understood that part of the thoracic aorta lying beyond the distribution of the main arterial trunks—between the left subclavian artery and the point of insertion of the ductus Botalli. I have recently gathered together four cases of isthmus stenosis at the Johns Hopkins Hospital, and reported them under the caption "Stenosis of the Isthmus (Coarctation) of the Aorta," etc.[1] I used this nomenclature because such cases are described as "Stenosis of the Isthmus" in every language in which I found them reported excepting the English language, into which the term "coarctation" has introduced a needless idiosyncrasy.

In contrast with the outspoken and unequivocal physical signs that are usually associated with stenosis of the isthmus of the aorta, the signs of generalized hypoplasia of the aorta are, at the best, somewhat obscure. In this condition there is no collateral circulation, which is the outstanding clinical feature of stenosis of the isthmus. Generalized hypoplasia of the aorta causes a so-called "critical phase" in the history of the circulation between the ages of twenty and thirty. Perhaps it can be said that cardiac hypertrophy and heart failure occurring in an individual in the third decade of life, without obvious cause, should make one suspect the presence of hypoplastic aorta. If the pulse is definitely reduced in size in addition to these signs, and if roentgenological examination of the aorta falls in line with the supposition, then it may be said that there is a probability of this condition being present. It seems to me, however, that diagnosis of generalized hypoplasia of the aorta is almost impossible during life; the most that I would be willing to say in a given case is that the case in question has typical findings

*Read before the American Climatological and Clinical Association in September, 1926.

[1]King, John T., Jr.: Arch. Int. Med., 1926, xxxviii, 38.

of hypoplasia of the aorta, and that the diagnosis of the same may be made with reasonable safety.

A case illustrating this condition occurred at the Johns Hopkins Hospital in January, 1926.

CASE 1.—B. H. A laborer, thirty-six years of age, unmarried, entered the hospital complaining of shortness of breath. The family history was unimportant as related to his present illness. His general health had always been good, and he had had the usual childhood diseases but no rheumatic fever or chorea. He had influenza eight years before admission. There was a questionable history of one attack of tonsillitis. He had had a neisserian infection six months before admission. There was no history of lues. He had been rather a heavy drinker for several years prior to his present trouble. The symptoms of which he complained began eight months before admission, at which time he had dyspnea, some precordial pain and generalized edema. Dyspnea occurred on slightest exertion. There was some orthopnea. He had not worked for several months prior to admission, and during this interval his edema and dyspnea largely disappeared.

When he was admitted to the hospital his temperature was 99.4°, respiration 20, pulse 76, blood pressure 124/86. He was thin, undernourished, but in no obvious distress. He was quite bald and looked older than his age. He showed a rather marked depression of certain of the sutures of the cranium. There were a few small palpable glands. Eyes, nose and ears were essentially normal but there were numerous dental caries and pyorrhea. Lungs were clear except for a few moist râles at the bases. Abdomen was normal. Reflexes were normal. He showed a slight prostatitis.

At admission Dr. Neighbors made the following note on heart: The heart is enlarged to the left. The left cardiac diameter measures 5 cm. in the first interspace, 7 cm. in the second, 8 cm. in the third, 12 cm. in the fourth and 13 cm. in the fifth. The right cardiac diameter is 3.5 cm. in the fourth interspace. The apex beat can be seen as an undulating movement in the fifth interspace, 12 cm. from the midsternal line. There is no marked enlargement of the aorta. The cardiac rhythm is normal, at the apex there is a snapping first sound; following the second sound a third snapping sound can be heard. The sounds at the base are normal. At the apex the apical thrust is quite marked in systole.

Examination of the heart by Dr. W. T. Longcope was as follows: "Marked enlargement of the heart, wavy impulse without accentuated heart sounds: Great mobility of the cardiac dullness on change of position, free movement of lung over precordium; lack of fixation of the sternum on respiration; no Broadbent's sign. Liver is palpable.

Small pulse. Absence of enlarged arteries with distention of veins. By teleoroentgenogram, a round heart with small aortic shadow compatible with percussion outline." Dr. Longcope believed that congenital hypoplasia of the aorta was possibly present.

Blood pressure determinations: Patient in the horizontal position, using the mercury apparatus, were as follows: Right arm 150/60, left arm 110/55, right leg 144/75, left leg 155/90.

My impression of the case was that in the absence of everything else, a congenital anomaly of the aorta must be considered as probable cause of the cardiac hypertrophy and myocardial insufficiency. On repeated examinations no murmurs were found over the heart. The radial pulse was constantly noted by different observers to be of

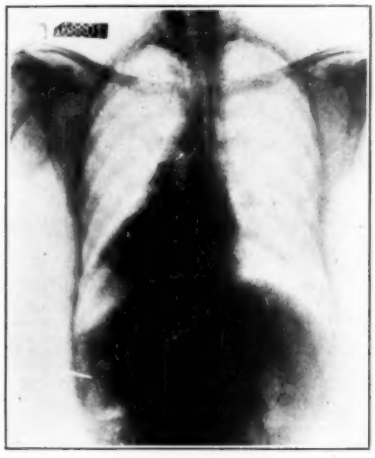

Fig. 1.—Case of probable hypoplasia of the aorta reported in this paper.

small size and volume. The actual size of the heart by teleoroentgenogram was 3.5 cm. to the right of the midline, and 11 cm. to the left.

On admission albumin was present in the urine, but it disappeared during the stay in hospital, and there were never any casts in the sediment; the urine was otherwise normal, except for leucocytes. Mosenthal kidney function test showed normal concentrating ability. Phenolsulphonephthalein showed 57 per cent elimination in two hours.

Blood examinations showed: R.B.C. 5,400,000 and Hb. 60 per cent on admission; R.B.C. 5,650,000 and Hb. 78 per cent on discharge. Leucocytes were between 9,000 and 11,000. Differential count was normal. Blood chemistry and blood Wassermann reaction were normal.

Electrocardiogram showed: rate 80, sinoauricular rhythm; P-R interval, 0.16 second; T-wave was inverted in Leads II to III. There was marked shift of the electric axis on change of position.

The teleoroentgenogram of this patient is shown in Fig. 1. It will be seen that there is definite cardiac enlargement, especially of the left ventricular curve, and that the aorta is, if anything, a little undersized.

The points of significance in this case are the presence of cardiac hypertrophy and heart failure without any evidence of valvular or pericardial or other cardiac disease. The pulse is of small size, and the x-ray shadow of the aorta shows no enlargement; furthermore, there is no hypertension. Except for the fact that this patient's symptoms developed in the fourth decade, and not in the third, the case is typical of hypoplasia of the aorta. In this case the blood pressure is higher in the legs than in the arms, which is normal.

I have not seen any references to blood pressure findings of these cases in which the pressures were taken in both arms and legs, though there are a number of such reports in cases of stenosis at the isthmus. I am obliged to Dr. W. T. Longcope for the invitation to see and to report this case.

The signs of stenosis of the aortic isthmus are well defined and permit accurate diagnosis during life. The collateral arterial circulation which develops to circumvent the stenosis in the upper part of the descending aorta is responsible for the most striking physical signs. These anastomoses connect the deep vessels of the neck with points below the stenosis through the dorsal scapular arteries (branches of the transversalis colli arteries), also through the internal mammary arteries to the epigastric arteries, and, in some cases, through superficial arteries that may be seen coursing obliquely across the back from the upper axilla downward toward the spine. The sign that instigated the diagnosis in our five cases was bilateral pulsation in the interscapular region, due to the greatly distended dorsal scapular arteries. These cases show an increase in pulsation in all the arteries of the upper extremities and a decrease in the legs and in the abdominal aorta. There is hypertension in the arms, and hypotension in the legs, where the systolic pressure, when it can be determined at all, is approximately equal to the diastolic pressure in the arms. Aortic insufficiency is present in probably half of the reported cases, and it occurred in two of the five cases we have seen. Systolic murmurs are audible over practically the whole of the anastomotic circulation, and there is a well-marked systolic murmur at the site of stenosis in the left interscapular area. There may be no cardiac hypertrophy, though the cases with complicating aortic insufficiency show it. The aorta in uncomplicated cases is usually slightly dilated proximal to the stenosis, especially near the point of constriction.

In addition to the four cases of stenosis of the aortic isthmus from the Johns Hopkins Hospital already reported, I have recently had

the opportunity of seeing a fifth case recognized by Dr. W. S. Thayer in his private practice.

CASE 2.—A young lady, single, thirty-three years of age, whose chief complaints were headache and dizziness, had been rejected for insurance because of hypertension.

There was nothing of importance in her family history. Among her previous illnesses were "typhoid pneumonia" at five, and scarlet fever and diphtheria at seven. Between the ages of sixteen and thirty she had had several attacks of swelling and soreness of various joints, none of which incapacitated her. Three years ago she had had a tonsillectomy, with some improvement. Her only previous cardiac symptoms were precordial pain several years ago and a distressing tendency to palpitation of the heart for a number of years. She had no especial dyspnea, no edema, and no nocturia. The patient gave a history of intermittent claudication in the legs on walking, also of headache and giddiness.

Physical examination showed good general nutrition and color. The pharynx showed a little chronic infection. There was evidence of an early pulmonary tuberculosis. Physical examination otherwise was negative except for the cardiovascular system. Dr. Thayer's examination showed the P.M.I. not sharply defined. By percussion the dullness was 9 cm. to 9.5 cm. from the midline in the fifth space, 2.5 cm. to 3 cm. to the right. No retromanubrial dullness. The sounds were best heard at the point of maximum dullness. First sound was strong, followed by distant, rather wiry, systolic murmur which appeared rather late, best heard in the fifth left space near the sternum where it was rather superficial and was increased, if anything, on deep inspiration. This murmur was heard at the base in both areas. Both second sounds were sharp, the second pulmonic perhaps a little more so. Normal sounds were heard in the carotids, loudly heard in the back and especially in the upper part, with greatest intensity over the first dorsal vertebra. There was normal venous undulation in the right side of the neck. There was a loud, long systolic murmur heard just under the left clavicle which diminished as one came down over the heart, to become loud again only at the fifth left cartilage. This murmur was heard much more feebly on the right under the clavicle, but was loud above the right clavicle. The second murmur at the apex was slight and far away and seemed to be produced at an area different from that at which the first sound was produced. Opposite the second dorsal spine and a little above there was slight pulsation visible and palpable very close to the spine. There was no palpable thrill. There was also a distinct pulsation opposite the first dorsal spine in the left supraspinous fossa about 3 cm. from the spine. Over these areas was heard a long, loud murmur with systolic accentuation

which at its points of maximal intensity lasted through systole and almost into diastole. It diminished rapidly in intensity as one passed downward but was heard through the back. There was a small superficial pulsating artery just below the scapula in the middle of the right back.

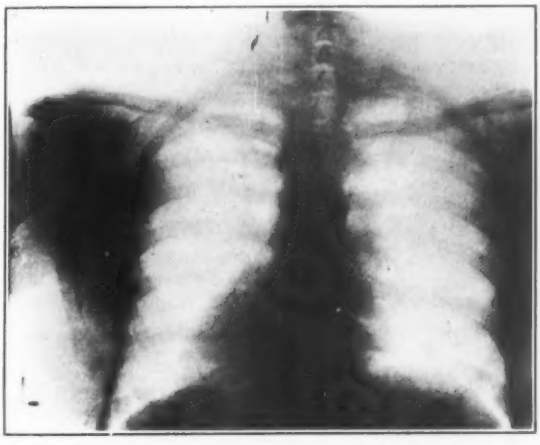

Fig. 2.—Case of uncomplicated stenosis of the isthmus of the aorta. Roentgen ray is not helpful in the localization of isthmus stenosis because of its location behind the cardiovascular stripe, and near the vertebral column. It is often necessary, however, to exclude aneurysm.

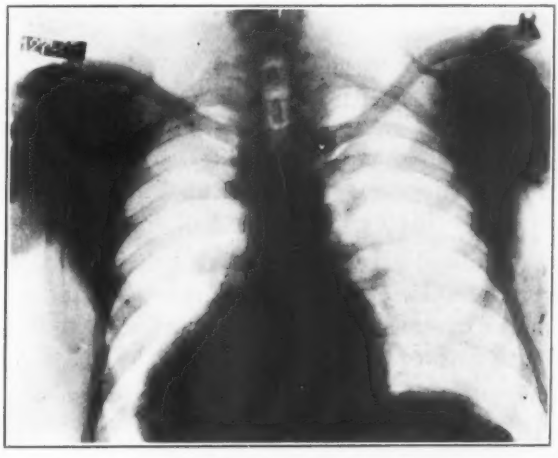

Fig. 3.—Case of stenosis of the isthmus with associated aortic insufficiency.

On examining this patient further I found blood pressure as follows: Right brachial 160/90; left brachial 160/100. In the right popliteal space pulsations were feeble but definite sounds were heard with pressure between 85 and 80; apparently the pulse pressure amounted to only 4 or 5 mm. No definite sounds could be heard on attempting to measure the blood pressure over the left popliteal artery.

There was a palpable pulsation in each popliteal artery though it seemed more feeble on the left than on the right. This pulsation was distinctly reduced as compared with normal. I could feel no pulsation in either dorsalis pedis artery nor in either posterior tibial artery.

Fluoroscopic examination: "The lungs on the whole are clear with the exception of little increase in right lower; a plate, however, has been taken of the lungs. The heart's contour is normal throughout and corresponds in a general way with percussion outlines since there was rather little shadow to the right and the shadow on the upper left contour is a trifle full. The aorta is not much dilated and it pulsates actively. Its size, however, impresses me as being more nearly that of a person in the fifties than of one in the thirties. There seems to me a very slight dilatation, especially in the descending part of the thoracic aorta."

Blood: Flows and clots normally, of normal viscosity, hemoglobin normal, about 90 per cent; R.B.C. of normal size and contour; no leucocytosis.

Urine: Light yellow, clear, 1.003, acid reaction, albumin negative, sugar negative, microscopic examination negative.

Phthalein output: First hour 50 per cent, second hour 12 per cent.

Basal metabolic rate: 2.5 per cent above the average for the patient's age and sex, indicating a normal metabolic reading. The resting pulse is 80.

Electrocardiographic report: The rate is 94 and quite regular; P-waves upright throughout. The P-R interval is 0.20 of a second. R-waves show a normal sequence in their heights. There is a definite slurring of the downstroke of the R-waves in the last two leads. QRS interval is not prolonged, measuring 0.07 of a second. The T-waves are upright in the first two leads, diphasic in the third.

Impression: Normal mechanism. The slurring of the R-waves is probably of no significance, although rather unusual. It is interesting that a similar observation was made in the case of S., one of the hospital patients with coarctation.

The nasopharyngeal examination and the sputum were essentially negative.

A few of the interesting points in this case are the presence of cramps in the legs (intermittent claudication), which occur only occasionally in patients with coarctation. This patient had no regular nocturia, whereas nocturia was a striking symptom in the hospital cases. She had the rather full aorta shown by these patients. The collateral circulation in this case was not very elaborate and was very similar to that in Case 4 of the hospital series; otherwise the case is quite typical and there seems to be no doubt of the diagnosis.

SUMMARY

A case is presented in which the signs of generalized hypoplasia of the aortic arch are present—cardiac hypertrophy without demonstrable valvular, myocardial or pericardial disease; myocardial insufficiency, small pulse and small aortic shadow by roentgenogram. This case is quite characteristic of hypoplasia of the aorta as described in clinical and pathological reports, except that myocardial failure occurred in the fourth decade, whereas the average age for the development of cardiac symptoms is in the third decade.

A typical instance of stenosis of the isthmus of the aorta is reported in a young woman. The chief signs were collateral arterial circulation, increase of pulsation and of blood pressure in the arms, and decrease of both in the legs, with characteristic murmurs. Cardiac palpitation, precordial pain and intermittent claudication in the legs were the chief symptoms.

A CASE OF DEXTROCARDIA WITH RIGHT (FUNCTIONAL LEFT) VENTRICULAR PREDOMINANCE, VENTRICULAR ECTOPIC BEATS, AND RETROGRADE CONDUCTION*

ROBERT H. POTTS, M.D.
AND
RICHARD ASHMAN, PH.D.
NEW ORLEANS, LA.

ONE of the most remarkable of anatomical abnormalities is *situs inversus*. Although not common, this condition is nevertheless frequent enough to have been noted many times in the clinical and anatomical literature.[1, 2] Thus a single typical case could hardly be regarded as of sufficient interest to deserve special report. Electrocardiographic studies of cases of dextrocardia with disturbances of the cardiac function are, however, rare,[3] so that the case herein reported seems of particular interest. Our case is exceptional in two respects: (1) The electrical axis during the time of inscription of the chief ventricular deflections of the electrocardiogram lies at approximately $-100°$ instead of at $+105°$ as is usual in dextrocardia. (2) Ventricular ectopic beats appear, some of which are transmitted in a retrograde direction to the auricles. This latter phenomenon is unusual even in the normally placed heart.

CASE HISTORY

Miss W. V., a stenographer, was referred to one of us by Dr. Thomas B. Sellers for a general medical examination.

In the *past history* there was nothing of note. She had been underweight as long as she could remember.

The *family history* likewise showed nothing of significance. Upon questioning it was found that she had a very capricious appetite and ate very irregular and insufficient meals.

The *physical examination* showed a tall, pale blonde girl of 24 years, with a rather listless appearance. Her weight was 99 pounds; height 5 feet 6½ inches. The chest was long and narrow in type; expansion was equal on both sides.

The heart was found on the right side, with a point of maximum impulse in the fifth interspace within the nipple line. No thrills or shocks were felt. A rather marked irregularity was noted, interpreted as due to extrasystoles appearing about every fourth beat. No murmurs; aortic and pulmonary second sounds about equal.

Heart rate, 110. Blood pressure, 90/65 mm. in both arms. The diastolic pressure was very difficult to determine because usually the only Korotkoff sound heard was at, or just below, 90 mm. Lungs, negative; liver dullness on the left side. Abdomen, negative except that the liver was just palpable; spleen, not felt; stomach resonance on the right.

*From the Departments of Therapeutics and Physiology, Tulane University.

Laboratory examination. Urine, 1018 sp.g. and microscopically negative. The blood, except for a moderate secondary anemia (erythrocytes, 3,670,000), was negative.

Basal metabolic rate, –10.

Roentgenogram. Transposition of the viscera. The bronchial tree of the left lower lobe was more or less thickened. Except for their transpositions, the topography of the heart and of the great blood vessels appeared to be normal. As was anticipated from the slender build of the patient, the heart's anatomical axis was inclined toward the vertical.

There is therefore nothing remarkable clinically about this case except the degree of underweight, the *situs inversus totalis*, and an irregularity of the heart.

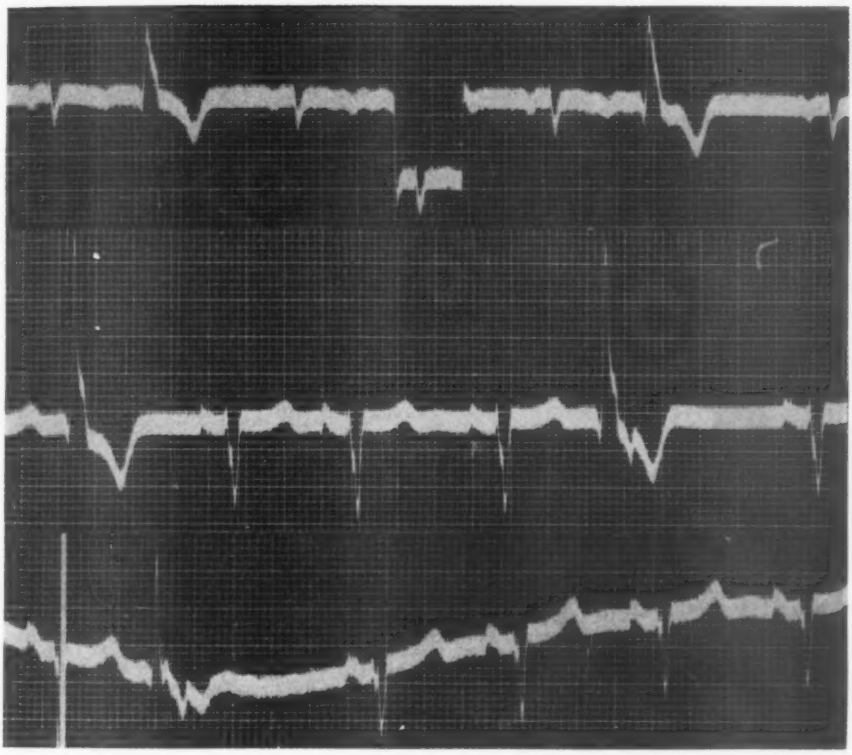

Fig. 1.—Leads I, II, and III from above downward. Usual wiring. Retrograde conduction after the second ectopic of Leads I and II, and after the single ectopic of Lead III. Time in 0.2 and 0.04 seconds.

THE ELECTROCARDIOGRAMS

In Fig. 1* are reproduced the three leads, taken in the usual manner. In confirmation of the physical findings and of the roentgenogram, Lead I presents the typical picture of dextrocardia[4, 5, 6]; the P-, R-, and T-waves, normally positive or upright, are here inverted. As anticipated, Leads II and III show positive P- and T-waves; but

*We are indebted to Dr. Upton Giles for the electrocardiograms of this figure. They were taken at the Baptist Hospital several days after we had obtained electrocardiograms illustrated in Fig. 2.

the R- and S-waves are quite unlike those usually exhibited in dextrocardia. Occasional ventricular ectopic beats interrupt the otherwise slightly irregular (sinus arrhythmia) rhythm; a few of these premature ventricular impulses are transmitted retrogradely to the auricles.

The ectopic ventricular beats and retrograde conduction.—There is nothing unusual abut the ventricular ectopics displayed by this case. They were present, but less frequent, when the heart rate was rapid (115 to 125), as it was on the first day electrocardiograms were taken; later, when the heart rate was slower (75 to 90), they were more frequent. On the earlier date they appeared late in diastole, so that they commonly barely anticipated the arrival of the auricular impulse; in fact in some instances (Fig. 2, Lead I) their form is modified

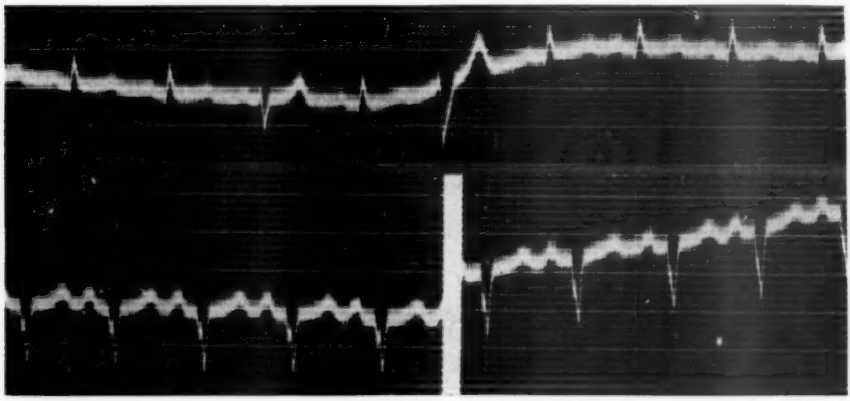

Fig. 2.—In obtaining these electrocardiograms, the arm contacts were reversed and the leg contact was on the right leg. Lead I, upper line, is therefore left arm—right arm; Lead II (first half lower line) is left arm—right leg; and Lead III is right arm—right leg. The third and fifth ventricular complexes of Lead I are ectopic. Note the altered form of the first ectopic caused by the almost simultaneous entrance of the supraventricular and discharge of the ectopic impulses. Time in 0.1 and 0.02 seconds.

because they arose just as the auricular impulse entered the ventricles.

More interesting because of its comparative rarity is the retrograde conduction of certain of the ectopic impulses. This is illustrated in Fig. 1. It is not always easy to be certain that apparent retrograde conduction of ventricular ectopics is actually such.[6] Our case is a particularly clear one and our interpretation is proved, we believe, by the following considerations: (1) The P-waves following the ectopics are exceptionally clear and sharply inverted in Leads II and III. The reader should compare the smooth down-slope toward the T-wave in those ectopic complexes where retrograde conduction fails with those complexes exhibiting it. (2) Retrograde conduction occurs only when, from the time relations of the auricular and ectopic

ventricular systoles, it is clearly possible for the retrograde impulse to have reached the auricles in time to have anticipated the discharge of an impulse from the sinus node.* (3) The only other reasonable explanation of the phenomenon under discussion, namely, that the ectopics may be of A-V nodal origin with retrograde conduction from that node and aberration of the impulses reaching the ventricle because of their prematurity,[6] is ruled out in this case since many of the ectopics are clearly not sufficiently premature to have caused such aberration.

The fact of retrograde conduction in our case is, we believe, good evidence that the conducting tissues are quite normal, not only because such conduction is possible but because of the brevity of the R-P interval, 0.15 sec., which is only slightly longer than the figure for the P-R intervals.

Dominance of the right (functional left) ventricle.—Since in a case of *situs inversus totalis* there is usually no disturbance of the anatomical relations of the various body structures,[2] except in so far as the individual is a mirror image of the normal, there is an *a priori* reason for expecting that the more thick-walled ventricle which supplies the systemic circulation will be found posteriorly and to the right, while the ventricle which discharges blood into the pulmonary artery will be anterior and to the left. The roentgenogram appears to confirm this conclusion. Consequently we shall call the first-mentioned ventricle the functional left; the other, the functional right ventricle in our subsequent discussion. As is well known, in the typical case of *situs inversus totalis*, with its associated dextrocardia, the electrocardiograms are precisely like those which would be obtained from a normal person after interchanging the contacts to the arms and leading from the right instead of the left leg. Changing the leg contact has in fact only a slight effect. As can readily be visualized, under these circumstances all deflections of Lead I (LA-RA) are reversed in direction; Lead II (LA-RL) corresponds closely to the usual Lead III; while Lead III (RA-RL) corresponds to Lead II. In a subject with dextrocardia but with an otherwise perfectly normal heart it is evident that these same transpositions of the leads will result in electrocardiograms of completely usual appearance. Such lead transpositions were made in our case, the electrocardiograms being reproduced in Fig. 2. The legend with that figure states the lead relations precisely. It at once becomes clear from inspection of this figure that this case is not normal; the electrocardiograms as they stand are those

*We wish to note the frequently encountered statement purporting to explain the failure to cause a ventricular response of the sinus impulse which is discharged almost simultaneously with the ordinary ventricular ectopic beat, namely, that the impulse, on reaching the ventricle, finds it refractory. Such a visualization of the event, unless it can be shown that the ventricular impulse is blocked on its retrograde path, is misleading. In perhaps the majority of cases of ventricular ectopics, as is clearly demonstrated by our clinical case, the actual event is a meeting and mutual extinction of the two impulses somewhere in the conducting system.

of marked left ventricular predominance or preponderance. Since the leads have been reversed to suit the transposed heart, the predominant ventricle is that on the right side of the heart, or, as we have called it, the functional left ventricle.

We have been unable to come to a definite conclusion as to whether we are here dealing with a preponderance or with an incomplete block of the left (functional right) bundle branch. There are several reasons for believing that there is no pathological lesion involving the bundle branch. (1) The comparative narrowness of the QRS group of deflections which is about 0.087 sec. in duration. Wilson and Herrmann[7] regard a relatively normal QRS interval as practically ruling out disturbances of intraventricular conduction. (2) The absence of an exaggerated T-wave and of a diphasic character of the ventricular complexes.[7] It is true that in Leads II and III these complexes are diphasic in character, but the T-waves are not exaggerated nor is the complex of Lead I diphasic. (3) The normality of A-V conduction, as well as of retrograde conduction, counts against the presence of bundle-branch block, since in the latter condition defective A-V conduction is frequent. (4) The absence of the clinical signs of myocardial damage likewise makes the existence of bundle-branch block less probable. (5) The constancy of the QRS interval despite marked changes in the heart rate (75 to 125) is additional and, we believe, strong evidence against a lesion of the bundle branch. If bundle-branch block is present it is clearly incomplete or partial, for otherwise the QRS would be greatly prolonged. In the majority of such cases however the QRS interval will increase with cardiac acceleration and decrease with heart slowing because of the shorter or longer rest intervals afforded the injured tissue. This fact is mentioned by Wilson and Herrmann[7] and has been observed by one of us in a clinical case of incomplete bundle-branch block with auricular fibrillation.

The picture of predominance of the functional left ventricle is so extreme and in such sharp contrast with the absence of hypertrophy demonstrable in the roentgenogram, that it was thought worth while to determine the course of rotation of the heart's electrical axis during the time of inscription of the QRS deflections. For this purpose four complexes in each lead, taken in the usual manner, and as nearly as possible at the same respiratory phase, were measured carefully under the low power of a binocular microscope. The four sets of readings in each lead were averaged. In order to synchronize the events in the three leads, the apices of the S-waves of Leads II and III were considered as simultaneous.[8] The average curve for Lead III was then subtracted algebraically from the curve for Lead II. The resulting curve followed closely the average curve for Lead I and enabled us to synchronize the latter with considerable certainty.

Fig. 3 represents the average curves and the rotation of the electrical axis. These are described in the legend.

If we reject a bundle-branch *lesion* as a probable cause of the electrocardiographic picture of extreme preponderance, and if the roentgenogram gives us no reason to attribute the findings to hypertrophy of the cardiac muscle or to rotation around an anteroposterior axis,* we are forced to inquire what the remaining possibilities are. Rothberger and Winterberg[9] reported that, in the dog, cutting of the anterior subdivision of the left bundle branch resulted in an electrocardiogram resembling that found in left ventricular hypertrophy; cutting the posterior subdivision produced the opposite picture. Even assuming that similar lesions will produce identical effects in man and that as conspicuous a picture of preponderance could thereby be produced as that presented by our case, yet several of the same arguments previously advanced against the presence of a bundle-branch defect will still apply. More pertinent perhaps in application to our case are the results of Herrmann and Wilson[10] and of Meek and Wilson.[11] The former investigators found by a comparison of the $\frac{\text{left ventricle}}{\text{right ventricle}}$ weight ratios, as determined at autopsy, with the electrocardiograms obtained shortly before death, that the electrocardiographic picture of preponderance was trustworthy only for hearts whose combined ventricular weights exceeded 250 grams. Smaller hearts sometimes showed a picture of predominance, although the weight ratios were within normal limits; or they sometimes did not give the expected electrocardiographic picture when the weight ratios demonstrated a definite left or right ventricular preponderance. Some of these exceptions were explained as due to rotation of the heart around an anteroposterior axis; in other cases a slight degree of bundle-branch block was suspected; in still other instances the authors postulated a slight delay in one or the other branch of the His bundle, attributed not to an organic lesion but to congenital differences in the architecture of the conducting system.

If we may assume, as seems highly probable, that the results of Meek and Wilson[11] upon the dog will apply equally to the human, we have an additional possible explanation of the exceptional findings of Herrmann and Wilson. Meek and Wilson found that not only will rotation of the dog's heart around its anteroposterior axis produce electrocardiographic pictures resembling preponderance of one or the other ventricle, but that similar results follow rotation of the heart on its long axis. Thus rotation of the heart around its longitudinal axis, clockwise, if the apical end of the heart be viewed, produces the picture of right ventricular preponderance; rotation in the opposite direction simulates left ventricular preponderance.

*Such an extreme picture of predominance could not be produced in this way within the limits of rotation possible within the thorax.[10]

In our clinical case it seems improbable that rotation around a longitudinal axis can alone account for the conspicuous electrocardiographic picture of predominance. If we assume in addition that, as a consequence of an unusual arrangement of the conducting system, the functional left ventricle is activated slightly earlier than the functional right, we may have a sufficient explanation of our findings. Fig. 3 suggests that the functional left ventricle is first activated and completely dominates the picture during the initial period of 0.025 sec. when the electrical axis is rapidly rotating in a clockwise direction from about $+60°$ to $-130°$; that during the next 0.01 sec., when there is a counterclockwise rotation, the impulse may be passing into the functional right ventricular apex; and that, from this time on, the predominant (because rotated) functional left ventricle gets the

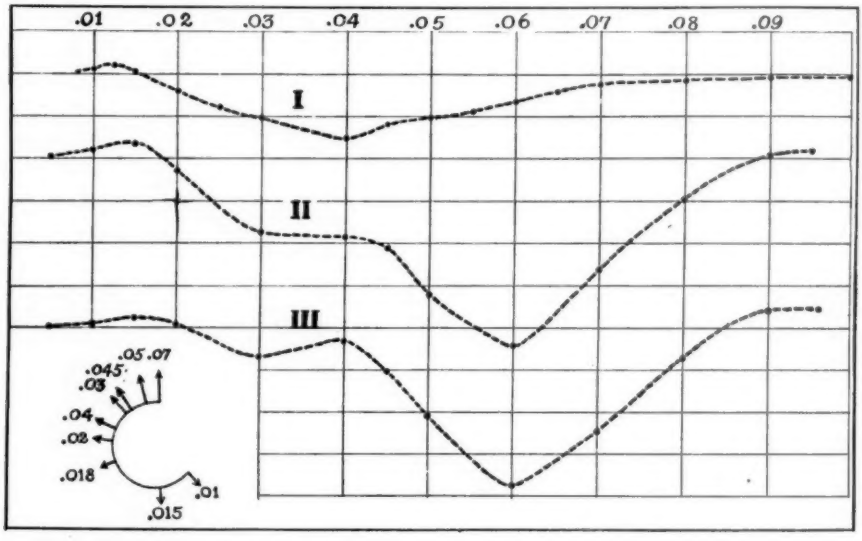

Fig. 3.—Each curve, Lead I, II, and III, is an average from measurements of 4 QRS complexes which are so plotted as to make possible a determination of the rotation of the electrical axis. The diagram at the lower left represents this rotation. The times at the end of the arrows correspond to the times as shown at the top of the figure; not, as will be seen, to the times from the beginning of systole.

upper hand and the clockwise rotation is resumed. This explanation leaves the extreme rapidity of the initial clockwise rotation unaccounted for, and any suggestion as to its cause, such as bridging of the left ventricular cavity with free strands of Purkinje tissue,[12] would be mere speculation.

SUMMARY

This paper presents an electrocardiographic study of a case of dextrocardia which is unusual in two respects. (1) The electrical axis during the time of inscription of the chief ventricular deflections lies at approximately $-100°$ instead of the usual $+105°$. This indi-

cates pronounced predominance of the functional left (systemic) ventricle. (2) Ventricular ectopic beats appear. Occasional premature ventricular impulses are transmitted in a retrograde direction to the auricles.

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PAROXYSMAL TACHYCARDIA IN INFANCY*

Report of Two Cases With Comments on the Differential Diagnosis

C. E. Colgate, M.D.
and
Hugh McCulloch, M.D.
St. Louis, Mo.

TWO cases of tachycardia in infants are reported because of the early age of the patients and because certain features of the electrocardiogram are not clear, making the diagnosis difficult. The absence of atrioventricular block, the fact that the attacks began and ended abruptly and were of short duration, and the fact that the infants seemed normal at other times, justify the diagnosis of simple paroxysmal auricular tachycardia. But since no electrocardiograms were obtained at the beginning or end of the attacks and since the cardiac rate and the form of the complexes shown in the electrocardiogram made from one of the cases raises the question of auricular flutter, the diagnosis cannot be firmly established. In any event, such instances of tachycardia are rare.

The first patient was a male negro child, twenty-four days old. He was brought to the St. Louis Children's Hospital because of an extremely fast heart rate. He was born in Barnes Hospital at the thirty-eighth week of gestation, labor having been induced because of the large size of the fetus, and the contracted pelvis of the mother. No anesthesia or "seminarcosis" was used during labor. The baby was normal at birth and weighed 3,420 grams. About one year before the patient's birth, the mother had had an induced abortion because of high blood pressure and other symptoms of toxemia of pregnancy. None of these symptoms was present at the time the patient was born. Wassermann tests on the mother had been negative on several occasions and a Wassermann test on the cord blood at the time of the child's birth was also negative.

The infant had seemed well until the day of admission to the hospital. At that time the mother noticed the rapid rate of the heart, some dyspnea and a disinclination to nurse. The child was brought to the hospital about 4:30 P.M., having been seen in the meantime by several observers, who had estimated the cardiac rate to be three hundred per minute. The only other positive findings were some increase in the respiratory rate and an enlargement of the liver, which could be felt almost at the umbilicus.

*From the Department of Pediatrics, Washington University School of Medicine and the St. Louis Children's Hospital.

An electrocardiogram, taken at the time of admission to the hospital (Fig. 1) showed the auricles to be beating regularly at a rate of two hundred and ninety-one per minute. The ventricles were responding at the same rate. The QRS complexes were normal in form, showing only a preponderance of the right ventricle. The P- and T-waves could not be clearly identified, so that a measurement of the conduction time was impossible. Vagus stimulation, both by direct pressure and reflexly through ocular pressure, failed to have any effect. The patient was watched continuously until about 5:45 P.M., during which time no change was noted in his condition. When examined again at 6:40 P.M., however, the pulse was regular and easily

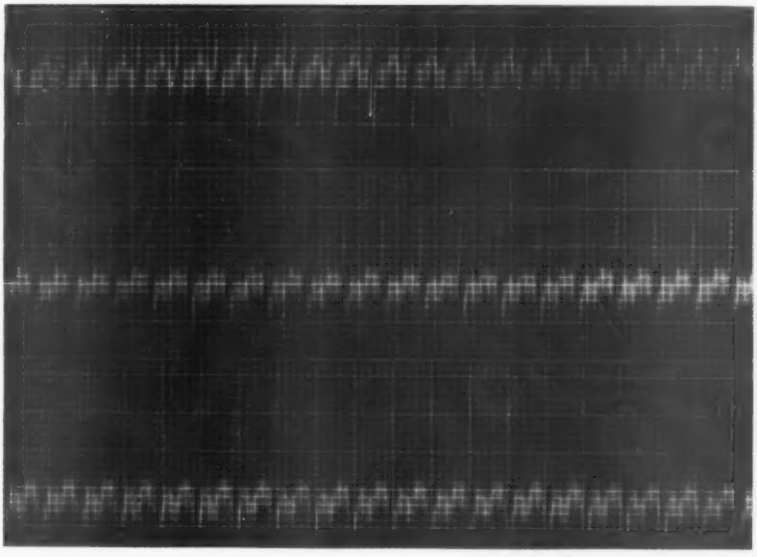

Fig. 1.

counted at a rate of one hundred and sixty. An electrocardiogram showed normal mechanism with a P-R interval of 0.08 second and a left ventricular preponderance. The next morning another record (Fig. 2) showed a similar condition, with a pulse rate of one hundred and forty. The dyspnea had disappeared and the liver edge was only one or two centimeters below the costal margin. No record was obtained of the beginning or end of an attack. Physical examination of the heart showed no abnormal signs at any time. The baby is now eight months old, has been seen from time to time and has had no recurrence of the tachycardia.

The case was considered one of paroxysmal auricular tachycardia, the youngest case so far which has been studied with the electrocardiogram, which survived the attacks and in which there was no

evidence of a cardiac lesion. The baby was two weeks premature and was first seen in an attack on the twenty-fourth day of life.

The second case occurred in the private practice of one of us (H. McC.). The patient was a white male child, born December 2, 1924. At the age of three weeks he had an attack of blueness. He was seen at this time by a physician, who found the pulse to be two hundred and fifty per minute. No electrocardiogram was made. An x-ray showed a normal heart outline. Following this there were frequent attacks, with each of which there was some distress, apparently abdominal, and slight cyanosis or pallor. The attacks began and ended abruptly, and lasted from one-half to six hours. When first seen by one of us on Jan. 27, 1925, the heart could be counted at

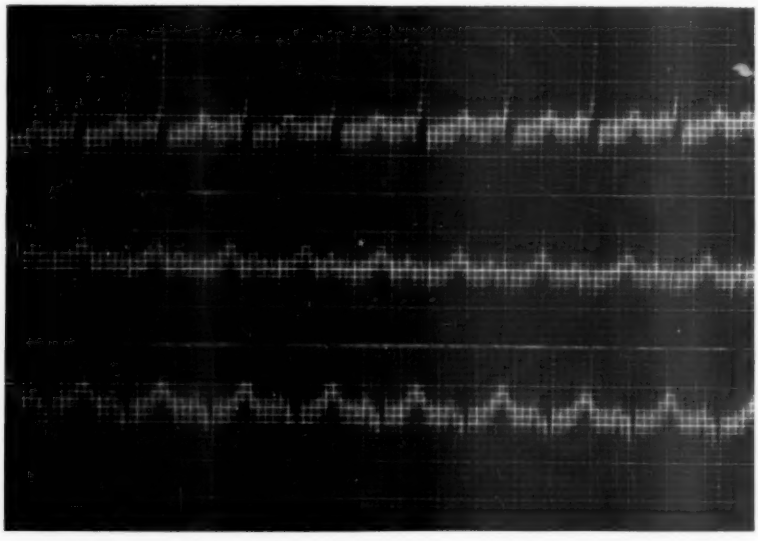

Fig. 2.

about two hundred and twenty and was quite regular. No abnormal physical signs in the heart could be made out either during or between attacks. There was present a simple, fast rate. No electrocardiogram was made. The last attack occurred when the infant was five months old. He is now twenty-one months old and seems to be a normal, healthy child. He has had several minor acute respiratory infections without cardiac disturbance. The family history is unimportant.

While no definite diagnosis can be made in this case because no electrocardiogram was made, the two cases seem similar in all respects.

DISCUSSION

Schuster and Paterson[1] have reported a case of frequent paroxysms of tachycardia in an infant nine weeks old. During the attacks the rate was "uncountable" and between attacks it varied from one hun-

dred and forty to one hundred and ninety. The patient died in a paroxysm and autopsy showed several congenital defects of the heart. No tracing of the heart action was made.

The youngest case on record was reported by Werley.[2] The attacks in his case began on the fourth day of life, and the child died when six days old. In this case, also, there was present a congenitally defective heart, as shown by autopsy. Polygraphic tracings were made at the time of the attacks.

Lewis[3] has reported a case of tachycardia in a child three months old, in which the rate, as shown by the electrocardiogram, varies from two hundred and seventy to two hundred and ninety. He calls this a case of flutter with 1:1 rhythm. Other cases in older children have been reported by Hutchinson and Parkinson,[4] Sutherland,[5] Koplik,[6] O'Flyn,[7] Thompson[8] and Amberg and Willius.[9]

Whether the cases reported here should be diagnosed as auricular flutter or paroxysmal auricular tachycardia is uncertain. The criterion by which these two conditions may be differentiated is by no means clear. Lewis[10] has arbitrarily limited the term "paroxysmal tachycardia" to those cases showing a regular auricular rate between one hundred and two hundred and twenty, while rates higher than two hundred and twenty he considers flutter; but cases diagnosed paroxysmal tachycardia appear in the literature with rates above two hundred and twenty. Other authorities consider the presence of some degree of A-V block to be the differentiating characteristic of flutter, but several cases,[3, 11, 12, 13, 14, 15, 16] of 1:1 rhythm in flutter appear in the literature, and there is at least one report[17] of three cases of heart block during auricular paroxysmal tachycardia. Of the cases of flutter with 1:1 rhythm, one[3] shows no other form of ventricular response. Others[12, 13] occur only at the beginning of an attack, giving way later to 2:1 or 3:1 rhythm and in others[11, 14, 15] the 1:1 rhythm was induced by exercise in the course of a paroxysm showing 2:1 or 3:1 rhythm. The 1:1 rhythm in flutter is more common in children,[14, 18, 19] presumably because of the shorter duration of the attacks and the greater responsiveness of the conduction system and ventricular musculature.

Another method of distinguishing flutter from paroxysmal tachycardia is the familiar one of producing atrioventricular block by either direct or reflex stimulation of the vagus. Levine[20] stresses this point. Wedd[21] says "the production of irregular ventricular block by vagal pressure in the neck will differentiate it [auricular flutter] from paroxysmal tachycardia, in which the vagus will either produce no effect or abruptly terminate the abnormal rhythm. But this procedure is often without influence in flutter and on one occasion . . . brought an increase in rate."

In 1912 Lewis[22] said, "It is not certain that the third type of tachycardia [auricular flutter] is strictly separable from the last [paroxys-

mal tachycardia].'' Others have held the same view, although Lewis has since recognized them as distinct clinical entities.[9] Keating and Hajek[23] consider that paroxysmal auricular tachycardia, auricular flutter and auricular fibrillation ''are one and the same condition, characterized merely by three different stages of varying degree and intensification,'' and this view is strengthened by the case reported by Parkinson and Mathias,[24] which they consider showed at first paroxysmal tachycardia with increasing rate finally developing into flutter with heart block. ''The close relationship among premature auricular beats, paroxysmal auricular tachycardia, auricular flutter and auricular fibrillation has long been observed and commented on,'' says Wolferth[25] who reported two cases of ''intermittent auricular flutter,'' the electrocardiogram from one of which showed, in his opinion, the close relationship among the first three. The similarity of the mechanism of flutter and fibrillation is already well known.

One is inclined to agree with Wedd[21] that ''a satisfactory definition of flutter is still wanting,'' and that ''an adequate definition must await the solution of paroxysmal tachycardia to differentiate flutter. For the present, flutter may be defined as a regular auricular tachycardia dependent on a circus movement.'' The possibility of premature auricular contraction, paroxysmal auricular tachycardia, auricular flutter and auricular fibrillation being varying degrees of the same process is very strong.

The prognosis in the cases here reported is necessarily uncertain, since no cause for the abnormality could be found. In the absence of other evidence of heart disease, however, and considering the satisfactory progress of both cases, we believe that the disorder is not likely to be dependent on a cardiac lesion and that the outlook is good.

SUMMARY

1. Two cases of paroxysmal tachycardia in infants are reported, with electrocardiographic records taken during and after the attack in one case.

2. The difficulty of deciding whether these are cases of simple paroxysmal auricular tachycardia or auricular flutter is pointed out, with some discussion of the methods by which these are distinguished and of the possibility of their being the same process, varying only in degree.

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THE HEART IN EMOTIONAL CONFLICTS*

SIDNEY I. SCHWAB, M.D.
ST. LOUIS, MO.

I HAVE used the term emotional conflicts in order to get away from the use of cardiac neurosis, because the latter implies too rigid an organization of symptoms. For the purpose of understanding and interpreting the condition which I shall attempt to tell about, a more elastic term is to be desired. That many of these disturbances are neuroses and share with them the usual mechanism is without doubt true, but by using a localized and single system too narrow a conception follows. Cardiac neurosis is a misleading term also because attention is focused on only one of the many avenues through which excess emotional activity is permitted to escape. Too much of the therapeutic effort, therefore, is directed to the heart and too little attention is given to the individual himself. The result is that the symptom complex becomes more and more conditioned, fixed, and the pattern conformity more permanently established. The term heart disease in the patient's mind, put there or not as the case may be, becomes a definite reality proved to be so by his own experience and observation. Emotional conflicts then explain the mechanism as far as it can be explained, and gives to these cardiac pictures a physiological background, which, it is believed, opens the way for a better understanding and a more direct, logical therapy.

In a previous publication called "Heart Disease in a Neurological Clinic" I have described the kind of cases of which the present paper treats and touched in some detail on their history, findings, and subsequent course. The material since then has increased, and much that was set down then has been confirmed by additional and repeated examinations. From an observational point of view there is not much doubt of the correctness of the clinical picture then drawn, but there is still a question as to the physiological explanation. On the point, however, of emotions arising out of conflict processes in the production of cardiac symptoms there appears to be ample evidence. In so brief an exposition as this, clinical histories and data of examination are out of place. They will appear in a future report. It is sufficient to sketch in outline the kind of cases falling under this class, to suggest an interpretation of the mechanism, and to trace a general line of therapeutic approach.

*Read before the Section on Nervous and Mental Diseases of the American Medical Association, Dallas, Texas, April, 1926.

From the Neuropsychiatric Division, Department of Internal Medicine, Washington University School of Medicine and Barnes Hospital.

A heart not the subject of demonstrable disease, either in its muscular, valvular or conduction mechanism, in an individual not the subject of disease elsewhere and in what may be called a normal body, is capable of extreme variations in its activities. The margin of excessive functions is striking. Such a heart may double its rate, may lessen its rate to some extent, it may become irregular, or it may show defects in timing. Some or all of the variations are a part of or may be a part of daily experiences.

There are three ways of changing the heart mechanism so that sensations of an uncomfortable kind will follow. These are matters of almost daily experience. One is by increased demands on the work of the heart, through muscular effort; the other is through emotional stimulation. The two may be combined and often are. The third is less easily defined because the stimulus is not easily determined. Certain processes, partly physical, partly chemical and to some degree mechanical, originate in metabolic processes, causing heart acceleration and thereby sensations of discomfort in and about the cardiac region. Such cardiac sensations follow the processes of this kind, and attention is directed to the heart as indirect or subsequent consequences. Two of the processes, that is, the first two, are played out in a state of awareness and are matters of repeated personal experience, thus ordinarily arousing no unusual or terrifying responses in the individual who is conscious of them. The third of these mechanisms is somewhat mysterious, as the impulses of acceleration are not produced by voluntary and objective effort and arise from no source that the patient knows of. For example, collection of gas in the large intestine producing abdominal distension may accelerate the heart beat, or the same thing may take place as a result of some chemical substance of unknown origin liberated in the chemical processes of digestion or other changes taking place in the body. Various combinations of these three are possible, and it is of no value to attempt to separate them out.

There are, then, three causative agencies which can affect the mechanism of the heart, and they are matters of observation, experience and experiment. One is effort; the other, emotion; the third, endogenous products formed within the body. The characteristic of all these three prime mechanisms is that whatever the process may be, the effect on the heart is incidental and temporary—when the stimulus is removed, changed, or altered, the heart takes up its normal formula of activity again, and its accommodation to the emergency demand lasts only a little longer than the emergency itself. The experience, no matter how manifestly disagreeable it may be, leaves no tendency behind it toward pattern repetition unless the specific stimulus is repeated. Through a series of like experiences the individual becomes accustomed to the cardiac phenomenon and regards the heart's temporary abnormal reactions as a normal consequence of effort, emotion, and to some extent

of uncomfortable sensations, particularly in the gastrointestinal system. In the observation of the normally altered cardiac action and the tendency toward return to a normal heart formula, the basis for the study of the establishment of an altered mechanism that is more or less constant is furnished. The factor or factors that produce the chronic alteration or the temporary alteration without observable cause is the problem on which this paper attempts to throw some light.

Of the three classes of causes which have been mentioned, one lends itself to study as something that may be of causal significance in the neuroses. It is that agency which affects the heart without visible or tangible evidence of its existence or even its reality. Obviously, this is emotion. What emotion is or how it acts is not a part of the present discussion, nor is it believed to be essential. All that is necessary is to accept that there is such a thing and that every one has evidence to that effect. Emotion is, let us say, an activity that can and does exist and can and does manifest itself without awareness in a specific sense and without visible, tangible or objective evidence. It is capable of what may be described as an unconscious dominance; that is, dominance without the state of awareness. The physical results may be matters of observation, and a group of internal sensations may be the only evidence of its activity. Therefore there is injected into the problem of causation a mechanism which is so detached from everyday observation or critical judgment that the individual is aware only of far-lying results and not of immediate causes. Compare, then, this state of affairs with tachycardia resulting from effort, or that which takes place after immediate emotional experience, or that which results in body sensations of a painful or disagreeable kind. It is evident as a matter of daily experience that emotion can produce cardiac symptoms, and if it is capable of doing the same thing without traces of its presence there is furnished a mechanism that is capable of various and widespread effects dissociated entirely from the ordinary data and demands of existence. It is something that is always present, an always potential activity irrespective of a logical demand for its use.

The supposition that emotion is capable of exerting its influence on the cardiovascular apparatus without an adequate, conscious stimulus similar in every way to its acute manifestation in the presence of an adequate conscious one is supported by numerous observations. It can be compared to the existence of a tonic muscular impulse in states of a resting muscle. Its existence is deduced from the fact that tone develops in a muscle which is volitionally moved, because the identical phenomenon is present in the two conditions—movement and rest. If emotion can produce all the evidence of a disordered heart when consciously present, and if the same phenomena result when the factor of awareness is not manifest, the conclusion is justified that there are identical stimuli—no new factor being added or evident.

Various descriptive terms are given to the emotional phenomenon associated with disordered cardiac function, such as fear, apprehension, anticipation and anxiety. These connote unstable states rather than specific things, and they are all variants one way or another of the most constant and primitive physiological emotion; that is, fear. For the purpose of this exposition the term emotion may be expressed by the term fear, and it is the fear reaction in respect to the cardiovascular system that is important. The response to fear on the part of the nervous system and its indirect effects on the respiratory, cardiovascular and gastrointestinal systems are matters of everyday observation and experiment. What is not so well recognized is that the cardiovascular response detached from objective or positive fear incidents belongs essentially to the same class of responses.

The word fear should imply here something that suggests perhaps dread; that is, there is an element of consequence, something that is suggestive of a future happening directly related to the immediate effect. I am seeking to broaden the meaning in order to explain or to suggest an element of continuance. The reason for this will, I trust, be clear in the final explanation.

The kind of case that belongs to this group is well known and has been recognized and described for a long time. The most outstanding clinical fact is perhaps the constant or almost constant tachycardia. This may be as high as that seen in exophthalmic goiter or in the rapid pulses of organic heart disease. Associated with this is a constant or almost constant sense of the heart as such. I cannot define it any more accurately than this. The term heart consciousness has been suggested. These patients are aware that they have a functioning mechanism in the left side of the chest producing sensation of a positively disagreeable kind. These sensations are not necessarily concerned with the beating of the heart. It is not movement or activity that is complained of, but definite sensations of weight, pressure, constriction, etc. The localization is not exact, nor can the subjective sensations be sharply outlined, nor do they correspond to the normal cardiac area. Out of these vague sensations develop or spring attacks of definite pain—often acute, sometimes terrifying, and again associated with all the qualities of typical angina. The distribution along the shoulder and left arm is not uncommon. That they follow the common anginal pathway is not the least striking of this manifestation. The rest of the picture is logically built up—respiratory increase, constriction of the chest, difficulty in breathing, pallor, sweat, tremor, vertigo and sometimes unsciousness. These are some of the manifestations. Palpation over the heart area shows often tenderness to pressure, and in some instances an area roughly approximating the cardiac outlines may be found hypalgetic to pin prick or light touch.

The functional disability of such cases is often marked. Loss of

strength, weight, incapacity to carry out former tasks, insomnia, loss of energy and interest, depression, etc., are often found or complained of. The clinical picture is that of a seriously sick person distressed by the conviction of a serious, definite and organic disease. It is a picture of heart disease in the common use of that vague term. Such cases examined with the most rigid care and with all the exact methods of a modern cardiac clinic show apart from the tachycardia no significant findings. Cardiographic tracings are practically all normal, basal metabolism figures are normal, and physical examination of the heart shows no evidence of valvular or muscular disease. Blood pressure is normal or slightly below normal; the proper relation between systolic and diastolic pressure is found. Such cases are referred to the neurological clinic showing no organic disease of the heart or vessels, or in many instances without important physical findings anywhere. They are called neurasthenic, irritable, or nervous heart, neurosis of the heart, or what not. What are they? Evidently they are habituated disturbances in the essential rhythmicity and the timing of the heart mechanism. The heart output is not altered, there is no tendency to muscle hypertrophy, and no suggestion of dilatation. There is then a chronic or almost chronic alteration in the pattern formula of the heart rate with the concurrent or associated symptoms that have been referred to.

Apparently what occurs is that there is freed a series of continuous impulses that so affect the mechanism of rhythm that there is a chronic increase in rate produced. This shows itself clinically as tachycardia. Most of the other symptoms are associated phenomena depending on two sets of things: one the result of the tachycardia itself, and the other the incidental psychical effect produced by the conviction that heart disease is present. These two sets of effects are constantly interchanging, so that clinically there is little opportunity to separate them out. There remains, then, the question of the mechanism which produces the increase in heart rate and the tendency on slight and often evanescent stimuli toward arrhythmia and rate fluctuations.

In cases belonging to the group under discussion, all common and many uncommon causes for tachycardia have been excluded by a series of examinations toward this end. It is necessary, then, to fall back on the extramaterial mechanism. This is associated with the most common of all heart accelerators—that is, emotion, physical effort not being a factor in the picture. It is an evident observation that most of these patients at the time of the attacks of tachycardia or in the course of a permanent tachycardia state are not aware of any source of fear or anxiety and cannot find in the experience of the moment any trace of fear except that which is associated with the phenomena of the heart itself. This fear and its accompanying

anxiety are logical effects of the abnormally acting heart as such and need not be considered apart from this. In seeking further for a mechanism that might act without awareness, the region of the unconscious presents itself. Is it possible that a more or less constant emotional flow exists that is bound up with the unconscious or unknown material represented by past, forgotten or even anticipated experience? The bed-rock of this whole question is whether emotion can, so to speak, become detached from experience in such a way that in seeking an outlet for expression it becomes shunted along the cardiovascular pathway and affects heart mechanism in the only way emotion can and that is by increasing its rate and disturbing its rhythm. As long, then, as these emotional conflict processes are present in the unconscious level, so long will there be streams of emotional effects which take as it were the facilitated pathway, producing such a break in the ordinary inhibiting mechanism that a new pattern of heart rate is created. This has the tendency to become the usual heart rate pattern exactly in the same fashion as does the cardiac mechanism of organic heart disease. In the latter there are tangible mechanical reasons evident through the examination of the heart muscle, through the vascular supply of the heart itself, and through the demonstration of indirect efforts on the heart due to toxic, mechanical or other sources. In the former, no such explanation is found.

The difference of method in studying these conditions is now apparent. It is essential to find out first whether there is a source of conflict, and then to establish the fact itself. Experimental proof is naturally lacking; controls are impossible. The proof of the correctness of this assumption can be found only if in a large series of such cases conflicts of an emotional kind can be demonstrated; if, after the conflict sources are discovered, adaptation to them can be made, and if after this there is a disappearance of amelioration of symptoms. The lessening of the tachycardia should be the first effect of the reduction of the emotional element in the conflict.

In a series of cases on which this paper is based, an attempt is made to answer this question. They are open to the usual criticism that is inherent in any attempt to solve a physiological problem without experiment and control. Apart from this they furnish evidence that a chronic tachycardia can be produced by emotional conflicts at the unconscious level and that the heart symptoms can be improved in some instances, cured in others, and not affected in others at all. The method of study is through analysis to find the sources, origins and nature of these conflicts. The more direct and less intricate the procedure is, the better will be the final result. The nature of these conflicts is the essence of everyday existence, representing all sorts of stresses, domestic, economic, sexual, or anything in short in which the

emotional life may be aroused to the extent that it cannot find adequate means of outlet into direct, effective and logical conduct.

The purpose of this paper, then, is to call to your attention a series of nonorganic heart cases chiefly characterized by tachycardia and its indirect consequences, caused by emotion as a result of conflict. These conflicts have escaped the awareness of the individual and produce their results because they are part of the unaware and automatic mechanism in which the controlling and directing power of emotion is lost or so disturbed that the outlet is through the cardiac mechanism which controls rate and rhythm, and not in purposive, intended, logical or effective conduct.

THE ACTION OF PITUITARY EXTRACT AND OF HISTAMINE UPON THE CORONARY ARTERIES OF THE TERRAPIN*

CHARLES M. GRUBER, PH.D., M.D.
ST. LOUIS, MO.

GRUBER and Roberts[1] have pointed out how important are changes in P_H upon perfusion rates of the coronary and cerebral vessels. They showed that adrenalin chloride produces dilatation of both the cerebral and coronary vessels because of its acidity. Likewise dilatation of the cerebral vessels is constantly seen after the injection of infundin (B.W.&Co.) and pituitrin (P.D.&Co.) which are also highly acid in reaction. In 1924 Sumbal,[2] investigating the effect of infundin upon the coronary vessels of the tortoise, apparently did not take into consideration changes in P_H in his experiments and he states, "The substance was injected into the tubing carrying the perfusion fluid to the heart in doses of 0.1 to 0.2 c.c. of the full strength solution, or of 0.2 c.c. of this solution diluted 100 or 1000 times." His results showed uniformly dilatation of the coronary vessels. It was found by the writer[3] that it required 1.5 to 1.56 c.c. of an N/10 NaOH solution to neutralize 1 c.c. either of infundin or of surgical pituitrin. Because of the effect of changes in P_H upon the coronary vasculature, the author has repeated this work using several solutions of different acidity.

METHOD

The heart was excised and suspended in such a way that the ventricular contractions could be recorded by a myograph upon the drum surface. The perfusion fluid was Ringer's solution, with a P_H of either 7.0-7.2 or 7.6-7.8 at room temperature, which varied in the different experiments from 20.5 to 27° C. but was constant for each experiment. It flowed through a glass cannula inserted into the coronary vessel and tied securely in place. The perfusion pressure varied from 40 to 100 cm. of water but was the same throughout each experiment. The fluid leaving the heart was collected by a funnel and the drops recorded by means of a receiving and a recording tambour. The time interval was in fifteen second periods.

The pituitary extracts used were, infundin (B.W.&Co.), pituitrin "Surgical" (P.D.&Co.), and pure pituitary substance (Dr. Maurice Smith's Extract[4]). The first two were injected in two forms, i.e., in the acid state as they occurred in the ampules and after the addition

*From the Department of Pharmacology, Washington University School of Medicine, St. Louis.

of N/10 NaOH until they had the same P_H as the perfusion fluid. Phenol red was used as the indicator in all cases in which the P_H was determined. In some cases, however, the phenol red was omitted and 1.5 c.c. N/10 NaOH was added to the infundin just before the injection. The pituitary powder was dissolved in the perfusion fluid and filtered before being used. All the injections were made through the rubber tubing directly into the perfusate.

Histamine phosphate (British Drug Houses) and ergamine acid phosphate (B.W.&Co.) were also used. They were dissolved in Ringer's solution and injected directly into the perfusate in doses of 0.1 to 0.2 c.c. of a 1:1,000,000 to 1:1,000 solutions. In order to determine how much of the organ was involved in each case a saturated aqueous solution of indigo carmine was injected into the perfusate before the cannula was removed. In two animals only the anterior half of the ventricle was perfused. In all the other hearts both anterior and posterior branches of the coronary were intact and perfused.

RESULTS

The results of my experiments fall into two groups (see Table I), according to the solutions used; those in which the pure commercial preparation was injected forming one, and those in which the P_H of

TABLE I

A table showing the effects of commercial pituitary extract, before neutralization and after neutralization, and of the aqueous extract of pure pituitary substance (Dr. Maurice Smith), upon the rate of coronary flow, and height and rate of ventricular contraction. The perfusion fluid was a Ringer's solution of either P_H 7.6 to 7.8 or °= P_H 7.0 – 7.2 at room temperature 20.5 to 27° C. The commercial preparations used were infundin (B. W. & Co.) and *pituitrin (P. D. & Co.). The quantity injected was 0.1 c.c. but occasionally †0.2 c.c. was injected. A total of fifteen turtles was used.

COMMERCIAL PITUITARY EXTRACT (ACID)					
RATE OF CORONARY FLOW IN DROPS PER MINUTE		HEIGHT OF CARDIAC CONTRACTION IN MM.		RATE OF HEART BEAT PER MINUTE	
Before	During	Before	During	Before	During
11	17	9	4	11	9
7.5	15	9	3	25	18
*†° 2	1	6	1	21	18
° 2	1-3	26	6	26	10
° 7	0.1				
10	11	10	3	32	32
†12	24	11	4	40	20
*†24	16-30	11	2	40	32
†15	28	7	2	44	32-36
26	16-28	20	11-18	46	24-42
6	2-8	9	5-9	44	8-30
12	8	16	9-11	36	34-44
8	5	8	4	42	24
14	14	11	9	38	38
°11	14				
°14	12				
° 7	0.5				
°10	7				
° 2	1				
†°33	28-37				

TABLE I—CONT'D

COMMERCIAL PITUITARY EXTRACT (NEUTRALIZED BY THE ADDITION OF N/10 NaOH)

RATE OF CORONARY FLOW IN DROPS PER MINUTE		HEIGHT OF CARDIAC CONTRACTION IN MM.		RATE OF HEART BEAT PER MINUTE	
Before	During	Before	During	Before	During
10	0.5	8	9	30	32
*†20	12.0	9	8	36	32
†12	0.5	8	5	32	32
25	16-30	15	15	44	42
28	8	14	12	32	28
32	16	14	14	24	24
†24	1	6	5	36	44
†16	1	13	9	24	28
†12	2	12	10-5	36	32
8	5	6	5.5	33	44
9	3	6	4.5	40	48
4	2	5	4.5	36	32
10	6	9	8	46	42

TABLE I—CONT'D

EXTRACT "MAURICE SMITH" PITUITARY SUBSTANCE

RATE OF CORONARY FLOW IN DROPS PER MINUTE		HEIGHT OF CARDIAC CONTRACTION IN MM.		RATE OF HEART BEAT PER MINUTE	
Before	During	Before	During	Before	During
10	4	9	11	20	12
13	5	9	11	19	16
4	6	10	15	36	36
4	1	10	13	33	24
4	2	9	12	28	20
5	1.5	9	10	25	20
15	0.5	8	8	24	16
25	3				
3	2	12	13.5	19	10
2.5	1.5	12	15	18	9
2.0	1.5	12	14.5	17	9
° 8	3				
°20	12				
19	6	15	11	39	31
16	12	11	10	36	36
20	16	10	12	40	36
29	19	24	23	33	18-43
8	5	10	11	32	4
5	1	5	6	33	0-4
12	10	10	10.5	44	50
7	5	4	6	38	40
°40	24				
° 8	3.5				

the commercial preparations was raised to the same value as that of the perfusion fluid, and in which pure pituitary powder was used, forming the other.

Acid Commercial Infundin and Pituitrin.—Either of these preparations, diluted or full strength, produced results such as Sumbal[2] got; i.e., vasodilatation of the coronaries, if the perfusion P_H is 7.6-7.8. If, however, the P_H of the perfusion fluid is 7.0-7.2 vasoconstriction of the coronaries is frequently observed even with these acid solutions.

Fig. 1 is a record selected from a large number of curves showing the effect of commercial infundin upon the coronary vessels and the

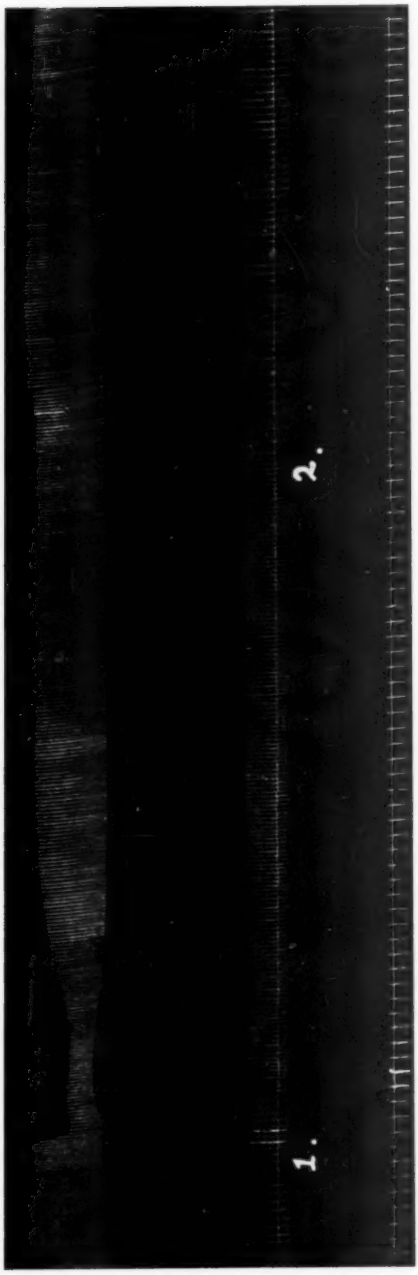

Fig. 1.—In this and the following records the top record is that of the contracting terrapin ventricle, below it the perfusion flow through the coronaries in drops and the bottom record is the time interval in fifteen seconds. Ringer's solution P_H 7.8 at room temperature. The perfusion pressure in this experiment was 65 cm. of water. *1*, 0.1 c.c. infundin; *2*, aqueous extract of pure pituitary powder.

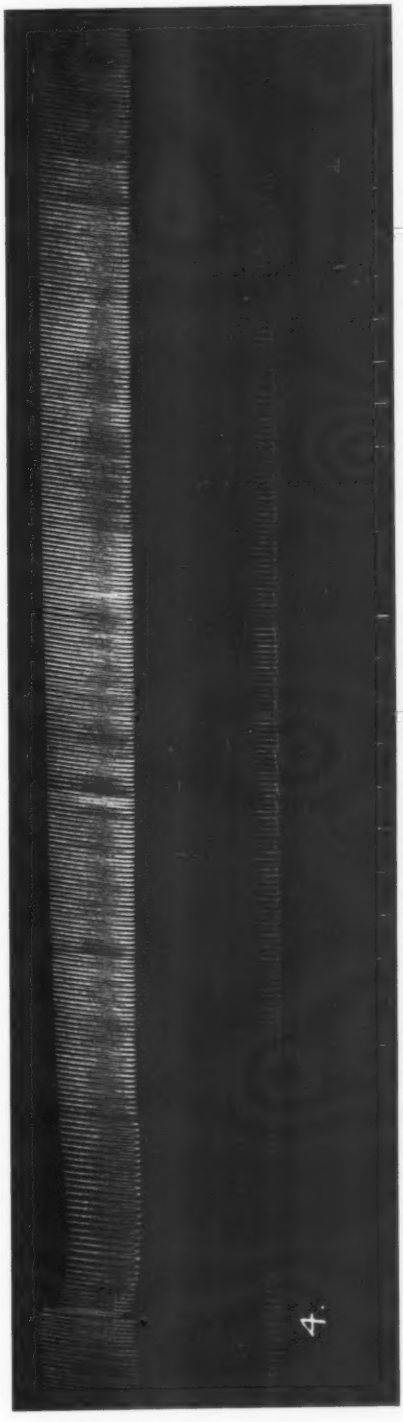

Fig. 2.—(See Fig. 1.) Perfusion pressure 50 cm. water. ½ 0.05 c.c. of N/10 HCl injected.

ventricular musculature. In this figure, at "1," 0.1 c.c. of infundin was injected into the perfusate having a P_H 7.6-7.8. The force and rate of ventricular contractions diminished almost instantly, with a simultaneous increase in the rate of coronary flow from 8 to 16 drops per minute. Such results were typical following the injection either of infundin or of pituitrin. It was also noted that the ventricle usually relaxes more than is normal.

N/10 HCl.—Experiments for the purpose of comparison were performed with the injection of N/10 HCl in small doses. In Fig. 2 at "4," 0.05 c.c. of N/10 HCl was injected into the perfusate. It will be noted that the results are similar to those observed in Fig. 1. There resulted shortly after the injection a marked loss in tonus, decreased

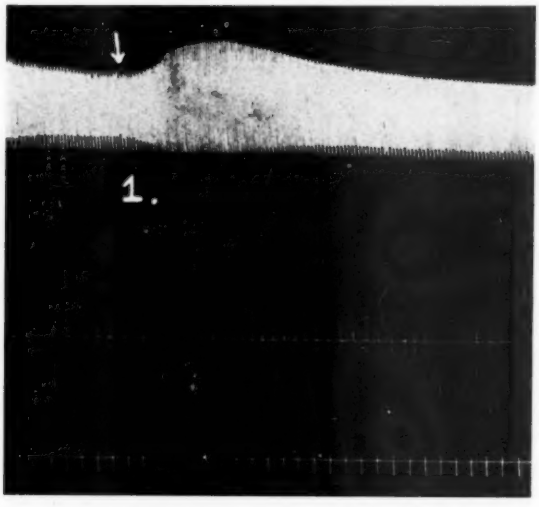

Fig. 3.—(See Fig. 1.) Perfusion pressure 65 cm. water. Room temperature. *1*, filtered aqueous extract of pure pituitary powder P_H 7.6-7.8.

rate and force of contraction, with a concomitant increase in coronary flow from 26 to 32 drops per minute. Here, as after the injection of the commercial pituitary extract, the dilatation lasted for some time after the passage of the acid through the coronary vessels. In both cases during the period of decreased ventricular activity the rate of coronary flow is diminished, a decrease probably of mechanical origin.

Pure Pituitary Powder Extract.—With but two exceptions (see Table I) the extract of pure pituitary powder gave coronary vasoconstriction. An increase in the force, but a decrease in the rate of ventricular contraction, was usually noted. Fig. 3 is one instance in which increased coronary flow was observed. There is such marked increase in the force of ventricular contraction that this increased flow is probably of mechanical origin. Fig. 4, is another record of the same heart as

was used in Fig. 3. In this case less marked change in ventricular contraction and vasoconstriction of the coronaries resulted. A similar condition is seen in Fig. 1 at "2." Here the injection was made after one of acid infundin, a marked decrease in the rate of coronary flow resulting after which the vessels remained in about the same state of tonus as they had been before the injection of infundin.

Infundin and Pituitrin Neutralized by the Addition of N/10 NaOH. —These commercial preparations, after neutralization, gave the same results as did the extract of pure pituitary powder. See Table I. It was also noted that this neutralized extract had little or no effect upon the ventricular contractions. In order to be sure that this change is not due to the addition of the indicator phenol red, more concentrated phenol red was injected into the perfusate; this had little or no effect.

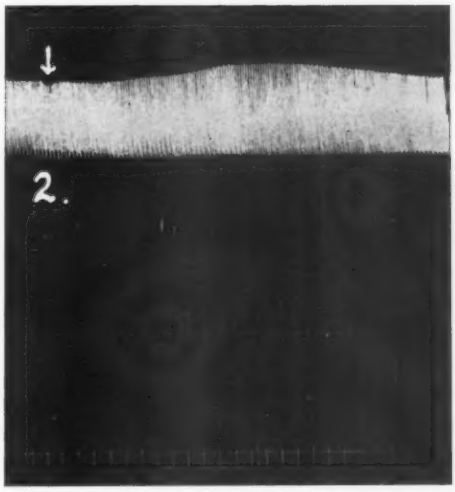

Fig. 4.—(See Fig. 3.) 2, Pure pituitary powder extracted with boiling Ringer's solution, P_H 7.6-7.8, and filtered.

See Fig. 5 at "3." Moreover coronary vasoconstriction resulted also in those cases in which 1.5 c.c. of N/10 NaOH was added to 1 c.c. of the commercial preparation, this amount having previously been found to be sufficient for neutralization, and in which therefore no indicator was used.

Figs. 5 and 6 are presented to show how the action of infundin differs, after the addition of N/10 NaOH to P_H 7.6, from the action of the acid solution seen in Fig. 1. In Fig. 5, at "3," 0.5 c.c. of Ringer's solution containing phenol red one drop per c.c., was injected into the perfusate. At "4," 0.1 c.c. of infundin to which had been added N/10 NaOH, P_H 7.6, was similarly injected, this injection resulting in coronary vasoconstriction without the depressing effect upon ventricular contraction noticed with acid infundin. In Fig. 6,

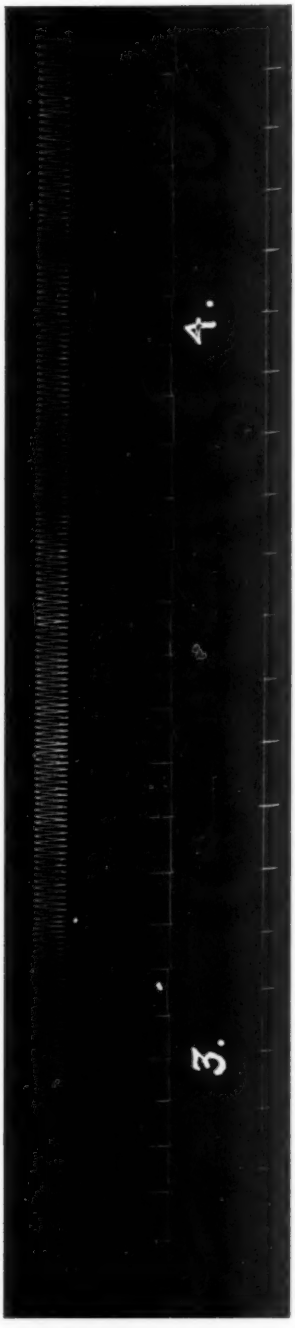

Fig. 5.—(See Fig. 1.) Perfusion pressure 40 cm. water. *3*, Ringer's solution + phenol red; *4*, infundin 0.1 c.c. + phenol red + N/10 NaOH.

Fig. 8 Continued.

TABLE II

A table showing the effects of histamine phosphate (B. D. H.) and ergamine acid phosphate (B. W. & Co.)* upon the coronary flow, and rate and height of ventricular contraction. The perfusion fluid was a Ringer's solution having a P_H of 7.6 to 7.8. The temperature was that of the room which varied on different days from 20.5 to 27° C. but was constant for each experiment. Number of turtles used, seven.

QUANTITY AND DILUTION OF DRUG INJECTION	RATE OF CORONARY FLOW IN DROPS PER MINUTE		HEIGHT OF CARDIAC CONTRACTION IN MINUTES		RATE OF HEART BEAT PER MINUTE	
	Before	During	Before	During	Before	During
0.1 c.c. (1:1,000,000)	9	7	6	6	32	32
"	12	5	7	4	44	42
"	51	27	14	12	38	38
0.1 c.c. (1:100,000)	20	15	12	6	32	40
"	28	22	15	10	20	24
"	8	4	6	6	28	28
"	6	4	7	7	42	42
"	14	11	11	11	38	38
"	48	24	14	14	40	38
0.2 c.c. (1:1,000,000)	47	4	14	12	22	32
"	39	28	12	12	32	32
"	27	6	13	12	31	26
"	14	4	11	11	22	20
0.2 c.c. (1:100,000)	37	12	12	12	32	32
"	7	4	6	6	36	36
0.2 c.c. (1:10,000)	8	1	11	8-10	40	38
*0.2 c.c. (1:1,000,000)	10	6	16	16	34	38
*0.2 c.c. (1:100,000)	10	4	11	11	38	40
"	18	8	13	13	12	32
"	46	8	10	12	24	30
*0.2 c.c. (1:10,000)	35	8	—	—	—	—

at "X" an equal quantity of infundin (neutral to the perfusion fluid) was injected into the perfusate of another heart. In this instance not only vasoconstriction of the coronaries can be seen but also an increase in ventricular tonus. In some hearts these injections not

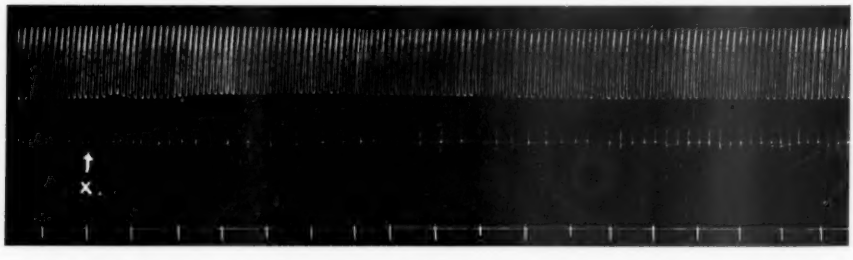

Fig. 6.—(See Fig. 1.) Perfusion pressure 60 cm. water. *X*, infundin 0.1 c.c. + phenol red + N/10 NaOH.

only increased the general tonus of the ventricle but established tonus waves to a very marked degree.

Histamine.—Sumbal,[2] in his report on the effect of histamine upon the coronaries of the tortoise, recorded only vasodilatation in the two animals studied. I obtained the opposite results. In seven terrapin 21 injections either of histamine phosphate or of ergamine acid phos-

Fig. 7.—(See Fig. 1.) Room temperature 27.5° C. perfusion pressure 80 cm. water. *1*, 0.2 c.c. of a 1:100,000 histamine phosphate solution injected. P_H 7.6–7.8.

phate a 1:1,000,000 to 1:1000 were made. (See Table II.) In every instance vasoconstriction of the coronaries was observed.

Figs. 7 and 8 were made on the same heart. In Fig. 7, 0.2 c.c. of a 1:100,000 solution of histamine phosphate was injected into the perfusate at "1," and in Fig. 8, 0.2 c.c. of a 1:1,000,000 solution of the same drug was injected, at "2." In Fig. 7, a slight increase in ventricular tonus and in rate of contraction, 24 to 30 beats per minute, are noted but in Fig. 8 no change in the ventricular tonus or the rate occurred. In both instances marked decrease in coronary flow resulted from the injection. Fig. 9 is another record taken from another animal in which 0.2 c.c. of histamine phosphate 1:1,000,000 was injected into the perfusate at "1." Increased tonus with decreased ventricular rate is observed. Before the injection the ventricular rate

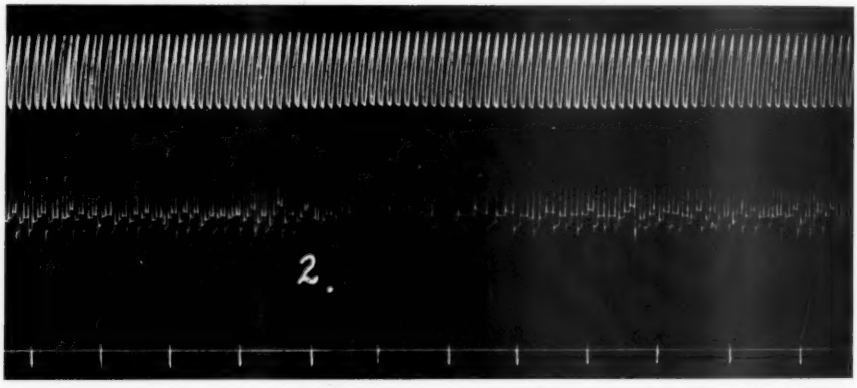

Fig. 8.—(See Fig. 7.) 2, histamine phosphate 0.2 c.c. of a 1:1,000,000 solution. P_H 7.6-7.8.

was 32 contractions per minute, after the injection it was 28. The rate of perfusion flow changed from 28 to a minimum of 5 drops per minute.

Ergamine acid phosphate, 0.2 c.c. of a 1:1000 solution, was injected into the perfusate at "1" in Fig. 10. Again marked vasoconstriction is noted. Inasmuch as the usual result of the administration of an acid is vasodilatation the effects observed must be due to the ergamine acid phosphate and not to changes in P_H of the injected fluid.

Adrenalin.—Amorphous adrenalin dissolved in Ringer's solution, P_H 7.8, and filtered was injected in a few cases. In all instances 0.1 c.c. was injected, the dilutions varying from 1:100,000 to 1:1,000,000. There resulted constriction of the coronaries, confirming the work of Drury and Sumbal[6] who used adrenalin chloride in doses of 0.2 c.c. of a 1:10,000 solution.

Fig. 9.—(See Fig. 1.) Perfusion pressure 60 cm. water. Temperature 25° C. *1*, Histamine phosphate 0.2 c.c. 1:1,000,000. Ph 7.6-7.8.

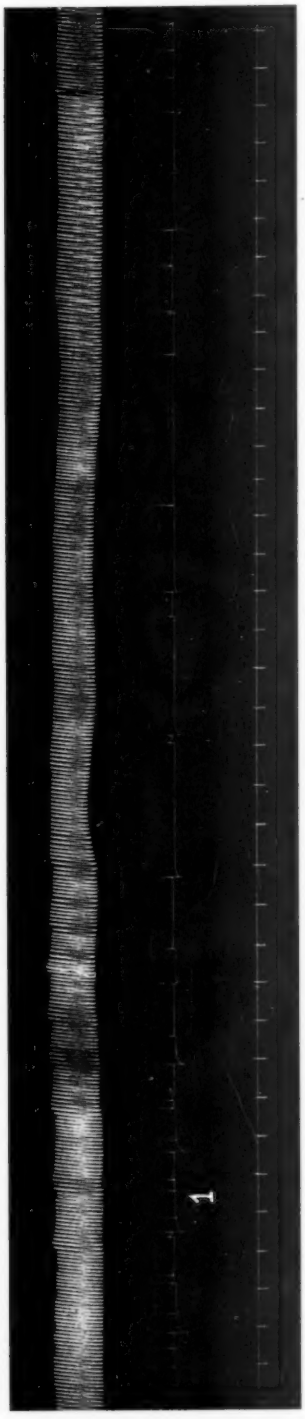

Fig. 10.—Ergamine acid phosphate 0.2 c.c. of a 1:10,000 solution.

DISCUSSION

My results with acid (N/10 HCl), infundin and pituitrin confirm the findings of Sumbal[2] with the same commercial pituitary extracts. However, the coronary dilatation observed is probably not due to the pituitary extract present but to the acidity of the injected fluid. When these commercial preparations are neutralized before administration dilatation of the coronary vessels does not occur, their action being similar to that of the extract of pure pituitary powder which is neutral in reaction. These experiments support Dale's[5] findings that pituitrin causes constriction of the mammalian coronaries.

My results with histamine phosphate and ergamine acid phosphate confirm Dale and Laidlow[7] and Krawkow's[8] findings on the action of this drug upon the coronaries of the rabbit.

SUMMARY

1. Dilute acid (N/10 HCl) when injected into the perfusate causes dilatation of the coronary vessels of the turtle.

2. Infundin and pituitrin, in the commercial state, are sufficiently acid to produce dilatation of the terrapin coronaries.

3. Raising the P_H of the commercial pituitary extracts infundin and pituitrin, by the addition of N/10 NaOH, to the same degree of alkalinity as that of the perfusate changes their action into vasoconstriction.

4. Extract of pure pituitary powder when injected into the perfusate of the terrapin heart results in coronary constriction.

5. Extract of the pituitary body causes constriction of the terrapin coronary vessels.

6. Histamine phosphate and ergamine acid phosphate cause constriction of the coronaries in the terrapin heart.

7. Adrenalin causes constriction of the coronary vessels (confirmation of Drury and Sumbal).

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VOLUNTARY PULSE CONTROL*

ALBERT S. HYMAN, M.D.
NEW YORK, N. Y.

VOLUNTARY and conscious control of the pulsations in the radial artery at the wrist, while not unknown, is sufficiently uncommon to merit interest. The ability of certain individuals to regulate or stop their pulse at will has been brought to my attention three times during the past year and since the available literature apparently records nothing in regard to this unusual ability to control what is ordinarily considered to be an involuntary cardiovascular phenomenon, a brief consideration of the problem may not be amiss.

Deliberate interference of the pulse at the wrist was known to the physicians serving in the Medical Corps of the Army during the World War. Soldiers in attempts to malinger were discovered to have used various ingenious devices in simulating disease of the heart. Recognizing that the pulse was used as a quick and simple method of determining the functional integrity of the cardiovascular system, they soon became aware of the fact that an irregularity in the pulse would retire them from active duty to hospital observation.

A method used with some superficial success was the placing of a rolled towel or any other soft globular object in the axilla and contracting the adductor muscles of the shoulder joint; the pressure thus exerted upon the axillary artery modified or entirely obliterated the pulsations passing through it in a manner not unlike that occuring in the Volkmann's paralysis syndrome. With a little practice, considerable skill could be developed in producing a small feeble pulse or an irregular pulse with no discernible effort on the part of the malingerer. A very simple examination, however, would usually promptly reveal this hoax.

Deliberate interference with the pulsations in the axillary artery has been noted in certain individuals with "loose joints"; in such persons extreme rotation of the humerus winds the artery around the bone with subsequent compression of the vessel. I have seen two men who could perform this feat. More theatrical is the performance of professional hypnotists who claim to control the pulsations in their radial arteries; after several deceptive sleight of hand maneuvers the arm is pressed firmly against the back of a chair or any other similar object until the pressure is great enough to obstruct the axillary artery.

*From the Witkin Foundation for the Study and Prevention of Heart Disease at the Beth David Hospital, New York.

Of more subtle mechanism is that wherein the pulsations in the radial artery are controlled by voluntary muscle contractions either consciously or unconsciously on the part of certain persons who possess unusual neuromuscular reflexes. It is a consideration of these cases which forms the subject of this paper.

The first case of this type was demonstrated to me through the courtesy of Prof. J. Rehfisch of the Polyklinik in Berlin. The patient was an ex-soldier who had become a professional entertainer and savant; one of his chief tricks was the ability "to stop his heart at will," evidence of which was given by permitting some physician in the audience to feel his pulse during the course of the experiment. After making several necromancic passes with his hands, the pulse at the wrist would entirely stop and he would apparently remain pulseless for fifteen or twenty seconds, after which the radial pulse would gradually return.

A brief examination of the patient showed that while it was true that the pulse at the wrist was no longer palpable, the heart sounds at the apex were unchanged either in rate or quality. Moreover, there was no change in the pulse of the opposite wrist nor in either of the femoral arteries. The origin of the constriction which was obliterating the pulse could be theoretically located at the left subclavian artery but the physiological and anatomical factors concerned in the phenomenon could not be studied because the patient refused to submit to a further examination.

The second case has been carefully investigated and studied by hemodynamic instrumentation through the courteous cooperation of the patient, Mr. J. R., a pharmacologist, fifty-one years old. He had noticed for some time that he possessed the peculiar ability "to will" his left pulse to become smaller; aroused by this unusual feat he continued to experiment until he at length discovered that his left radial artery would entirely cease pulsating if he "willed it hard enough." In a public demonstration before a group of more than one hundred physicians he succeeded in stopping his radial pulse for about fifteen seconds without appreciable effort.

A subsequent examination was made at the Heart Station of the Witkin Foundation. In this case, as in the German soldier, the stopping of the radial pulse was not associated with any change in the rhythm of the heart or in the pulsation of any other artery. Simultaneous electrocardiographic studies and polygraphic tracings of the left carotid and left radial arteries were made. (Fig. 1.)

It will be noted that while the radial pulsations stop, the electrodynamic cycle of the heart remains unchanged and the propagation of the carotid wave is unimpeded. Careful scrutiny of the "blocked" radial pulse shows, however, very small beats coming through (Fig. 2,

Fig. 1.—Simultaneous electrocardiographic (Lead II), left carotid, and left radial tracings. "Blocking" of the radial pulse shows no associated change in the carotid pulsations. There is also no change in the electrodynamic phase of the heart beat with the exception of a few small di-phasic waves which accompany the beginning and the end of the "blocking" of the pulse; these small waves are probably due to extracardiovascular muscle movements at the time when the artery is constricted and released, as they do not occur while the pulse is unchanged.

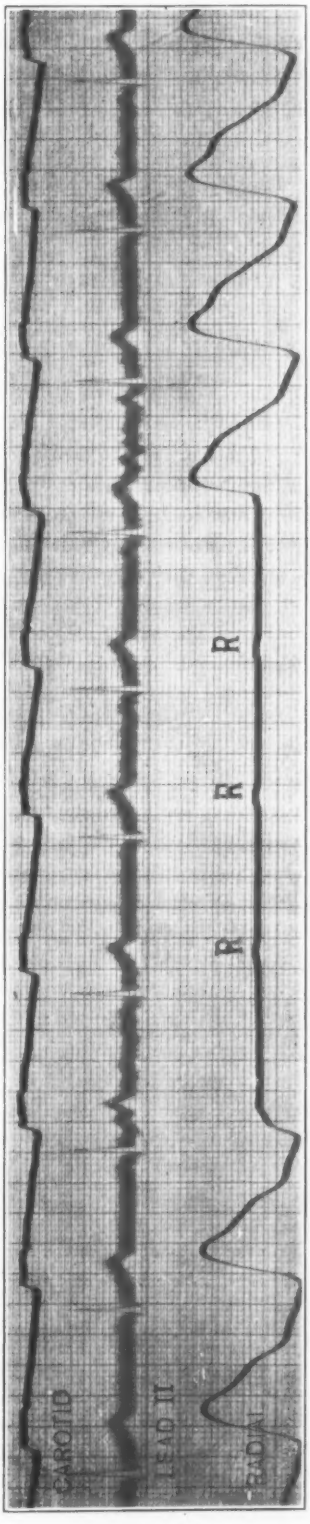

Fig. 2.—Simultaneous tracings as in Fig. 1. Note at *R R R* very small pulsations coming through the radial artery, due, perhaps, to incomplete compression of the subclavian artery during this experiment. These small beats cannot be palpated at the wrist and the impression is given that the patient is pulseless.

RRR), of too little amplitude to be palpated at the wrist but large enough to be photographed when magnified.

The unchanged carotid pulse and the transmission of the obstructed waves[1] would place the anatomical location of the "block" in the left subclavian artery where it passes over the first rib and under the insertion of the scalenus anticus muscle. While no muscle contraction is apparent during the course of the experiment, examination under the fluoroscope shows a rather small movement of the soft tissues lying under the outer end of the clavicle. The patient, himself, is not "aware" of muscle movement but he soon experiences a muscle fatigue which is indefinitely localized in the deep neck muscles of the posterior group.

Two anatomical explanations are possible: the first, that the subclavian artery perforates the scalenus anticus muscle[2] and because of its peculiar development this muscle exerts a sphincter-like action when contracted, shutting off the pulse wave in the artery. The second possibility is that the left subclavian artery has an unusually well-marked isthmus[3] which is readily constricted when the scalenus anticus muscle in contracting presses it against the tubercle of the first rib.

The longest period that the patient could "block" the radial pulse was 18.6 seconds; the ability to continue the control of the pulse was gradually lost so that at the end of an hour's experimentation he was unable to "block" the pulse more than a few beats at a time.

The position of the left arm was varied during the experiment; no change was noted until it was held at the level of the shoulder and then the ability to control the axillary pulsations was lost. However, if the patient was lying down, the pulse could be controlled even if the arm was held vertical.

Blood pressure experiments with the Pachon oscillometer were interesting; oscillations were normally recorded from the brachial artery as high as 145 and as low as 48 mm. Clinical blood pressure levels were established as systolic 138 and diastolic 68. During the experiment when the radial pulse was obliterated oscillations were at first recorded as high as 165 and then there was a gradual fall to 40, but even though the radial pulse was not palpable oscillations were readily recorded. This observation opens a fertile field of speculative inquiry, since it apparently sheds some light upon the problem as to the medium in which the pulse wave is propagated.

General physical examination of the patient was essentially negative and he is, to all intents and purposes, in excellent health. Attention can thus be focused upon the anomaly which he possesses without considering any other factor; he therefore presents an uncomplicated picture of so-called pulse control by unusual neuromus-

cular ability, and no consideration of "unexampled power of the will to control vital functions" need be discussed.

The third case is that of a young girl, Miss B. C. F., a college student, twenty-two years old, with "spiritualistic inclinations." As a child she had repeated attacks of tonsillitis and two distinct attacks of rheumatic fever with subsequent aortic-mitral disease. During the course of a routine cardiovascular examination it was noted that the pulse in the left radial artery was smaller than that in the right but another examination made only a few moments later to confirm the observation disclosed no discrepancy in the two radials.

Inquiry into the phenomenon revealed the fact that the patient since childhood had held an "unconscious fear" whenever a physician had taken her pulse; it apparently recalled to her many months when she had lain bedridden with cardiorheumatic affection and when the palpation of the pulse had such dire significance. She confessed that so extreme had been her childhood impressions that the mere act of feeling her pulse always was associated with an irresistible desire to stop her heart entirely and she learned that as a matter of fact she *could* stop her pulse if her "religious-mental attitude were proper."

So much of the patient's psychological background is related in order to demonstrate how confusing an unconscious neuromuscular act may become, more especially so when an anomalous artery and muscle relationship apparently exists. Since the muscle movement necessary to obliterate the radial pulse was "unconscious" the phenomenon could be elicited only with some difficulty and perseverance. The patient did not or could not entirely cooperate but a suitable graphic record was at length obtained. (Fig. 3.)

Examination of the record discloses that, as in the two previous cases, "blocking" of the radial pulse is not accompanied by any simultaneous change in the electrical phase of the cardiac cycle. While no carotid pulsation could be recorded in this case, on physical examination it could be readily demonstrated as remaining unaffected by the experiment. Of considerable interest, however, was the determination of the "latent period" required "to will the artery to stop"; this is recorded as X and Y and occupies the space of about one beat.

Inasmuch as the act was not entirely voluntary, the longest period over which the radial artery was "blocked" was 7.8 seconds. The ability to control the pulse was rather quickly lost; after four attempts on one day and three on another, the control disappeared. This may in part have been due to an extreme fatigue which the patient complained of during the course of the examination. In this patient as in the others the site of the arterial obstruction is probably in the subclavian artery with the relationships previously described.

Pulse control of the type illustrated by these three cases must be sharply differentiated from that due to extreme vagotonia in which

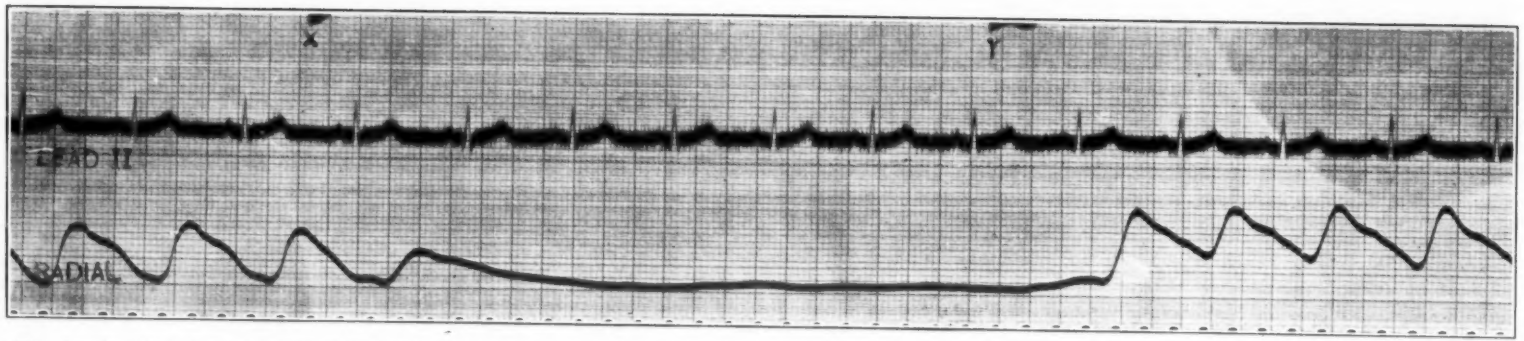

Fig. 3.—Simultaneous electrocardiographic (Lead II) and left radial artery tracings. At *X* the patient "willed her pulse to stop" and at *Y* she "released her will." It requires about the duration of one beat (0.75 seconds) to control the pulse.

the heart may be slowed as low as forty-four beats per minute when the breath is held. In this condition, while the pulse is slow, it does not stop and the entire cardiovascular system exhibits the same hemodynamic phenomenon.

SUMMARY

1. Three cases of so-called voluntary pulse control, with graphic records of two, show that, apparently as a result of anatomical anomalies or because of unusual muscular enervation, certain individuals possess the ability to obliterate the pulsations in their radial arteries.

2. Graphic records clearly demonstrate that the phenomenon is not due to any change in the cardiac cycle but that the subclavian artery is apparently constricted by a sphincter-like action of the scalenus anticus muscle or other muscles of the scalene group.

3. These cases are interesting in that they may be confused with true cardiovascular affections and it is undoubtedly possible that many persons may unknowingly possess the ability to alter the character of their radial pulse.

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THE INCIDENCE OF RHEUMATIC HEART DISEASE AMONG DIABETIC PATIENTS

JOSEPH H. BARACH, M.D.
PITTSBURGH, PA.

AS INTRODUCTORY to the present discussion, I wish to reiterate certain generally accepted facts concerning rheumatic fever and valvular disease of the heart. The first is that in more than 50 per cent of patients with rheumatic fever, there is a complicating endocarditis—the actual percentage being higher in the young. Secondly, that in the great majority of endocarditis cases, a history of rheumatic fever, chorea, quinsy and severe tonsillar infection, is obtainable. These statements need no further discussion here.

THE ALL-DISEASE CURVE

During the past year, I have been making a study of the relationship between the acute infectious diseases which an individual has experienced in early life, and the chronic disease with which he later presents himself for medical advice. Having this in mind for some time, I was especially careful in eliciting the history of these patients. The material for this study is taken from my private records. Out of the data obtained, I constructed first an "All-Disease Curve." This "All-Disease Curve" is based on the history of 1530 patients with various diseases as shown in Chart 1, and it illustrates the frequency of the preceding infectious diseases in a mixed group of patients.

THE VALVULAR-DISEASE-HEART CURVE

For the purpose of our present study, I superimpose upon this "All-Disease Curve," a curve based on 382 cases of valvular disease of the heart taken from my records. This chart shows that the "Valvular-Disease-Heart Curve" corresponds to the "All-Disease Curve" in all but two instances. The first deviation is found in the fact that in this special group of heart cases, tonsillitis was 25 per cent more frequent than in the general group. The other divergence in the "Valvular-Disease-Heart Curve" is to be found in the number of cases of rheumatic fever. Whereas in our mixed group of 1530 patients there were 156 cases of rheumatic fever, about 10 per cent, in the 382 cases of valvular disease, a history of rheumatic fever was obtained in 170 patients, about 50 per cent. These findings seem to coincide well enough with generally accepted ideas about the relationship between tonsillitis, rheumatic fever and valvular heart disease, and therefore affirm the validity of our charts and curves.

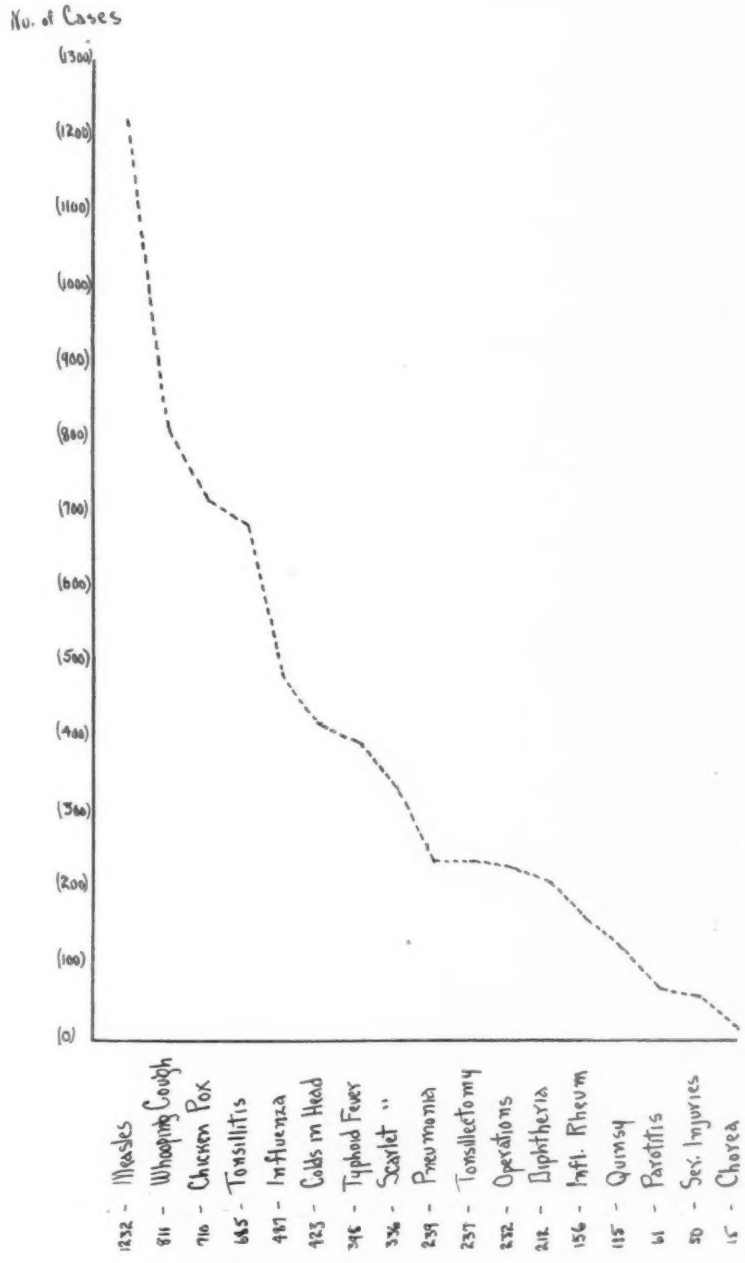

Chart 1.—All-disease curve—1530 cases.

THE DIABETIC CURVE

Chart 3 indicates the frequency of rheumatic fever in 226 diabetic patients. The curve shows that in point of frequency, rheumatic fever is neither more nor less frequent in diabetics than in nondiabetics; and

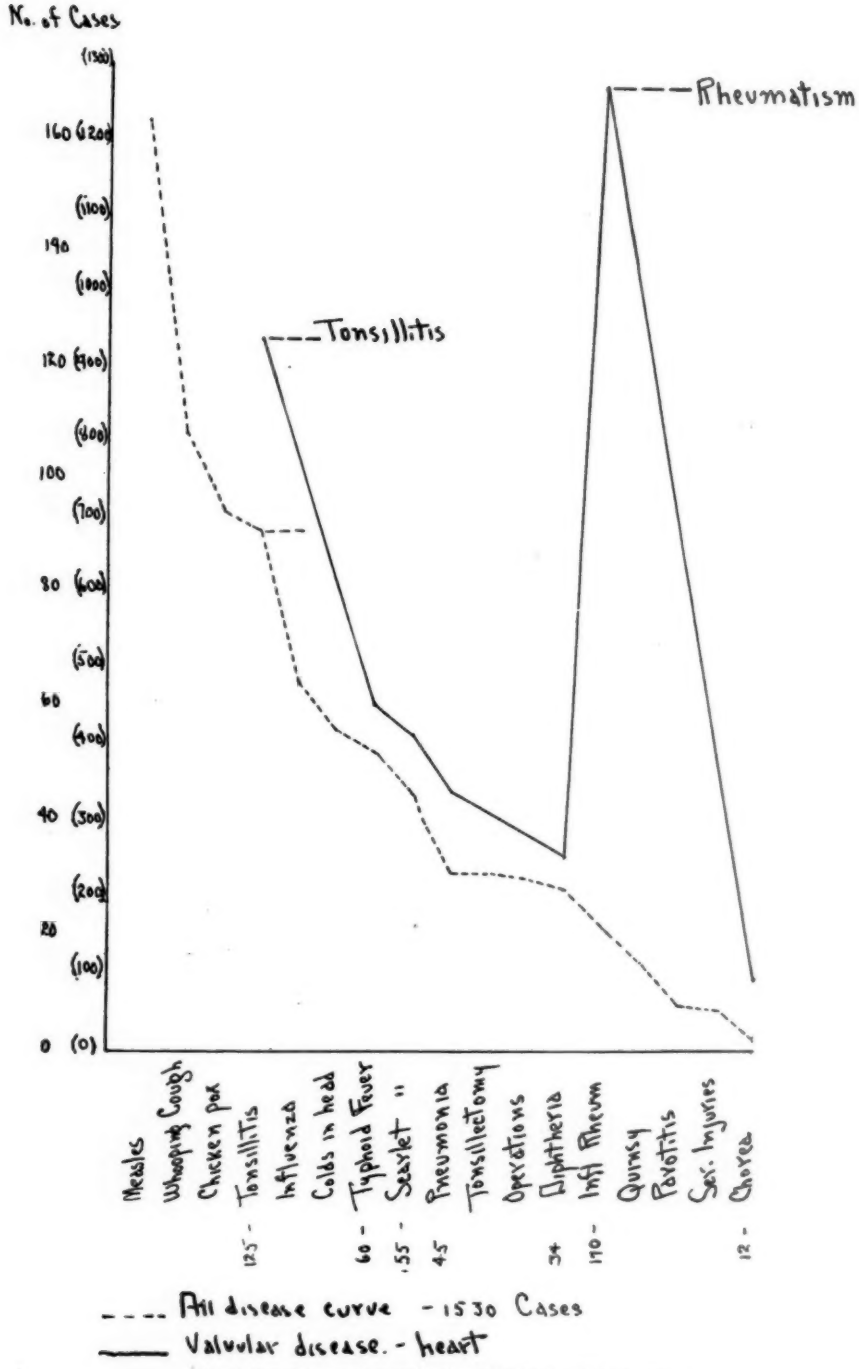

Chart 2.—Curve for valvular disease—382 cases.

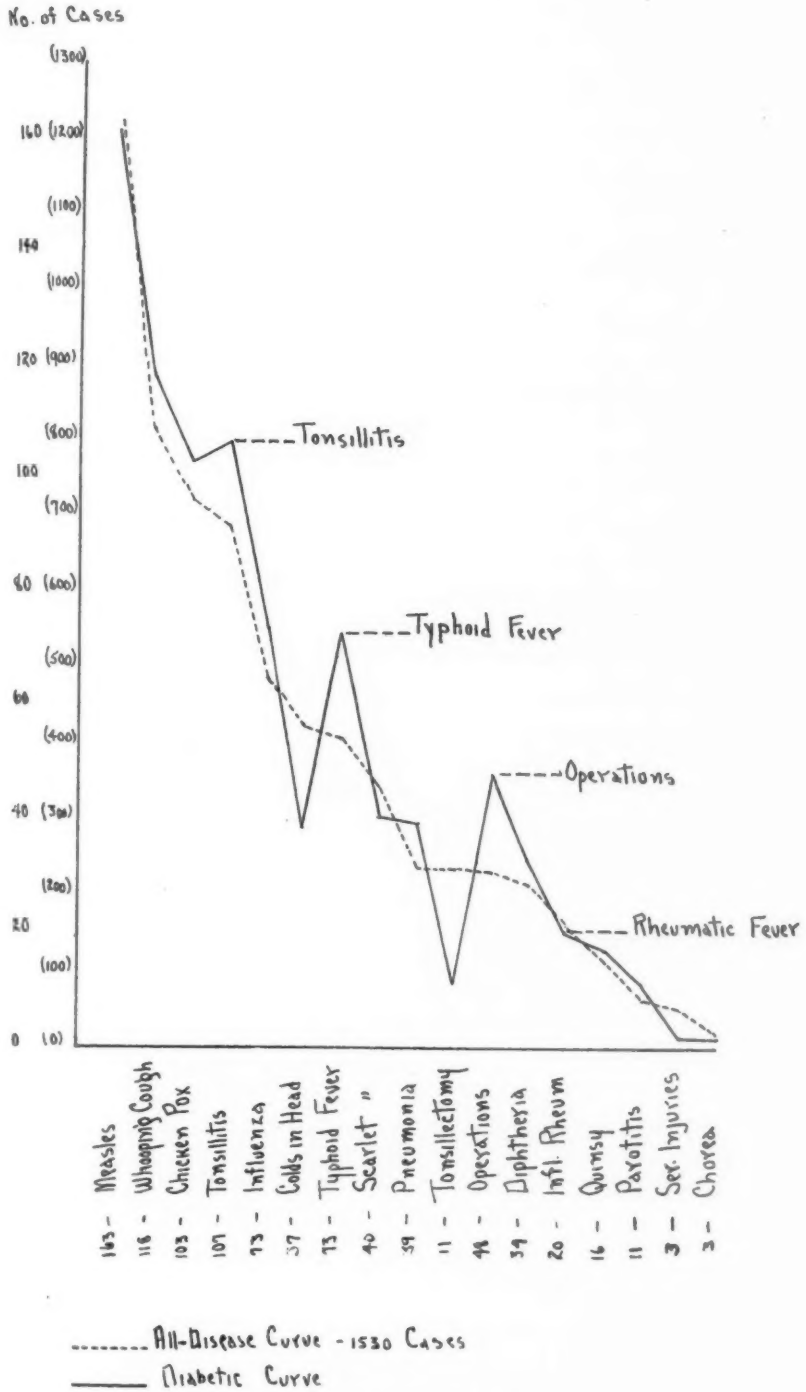

Chart 3.—Diabetes—226 cases.

it also shows that in both groups rheumatic fever occurs oftener than quinsy and less often than diphtheria.

In my larger group of 350 cases of diabetes, a history of rheumatic fever was obtained in 37 patients. These 37 cases of rheumatic fever occurred prior to the development of diabetes. In no case have I seen acute rheumatic fever in a diabetic. In 3300 diabetics, Joslin[1] records rheumatic fever in two cases only. Other than the comments of Joslin on this rare coincidence, I have been able to find no satisfactory references in the literature.

TONSILLAR DISEASE AND DIABETES

Chart 3 shows that in 226 cases of diabetes, a history of recurrent tonsillitis was obtained 107 times—about 50 per cent. In 1530 "all-disease patients," a history of tonsillitis was obtained 685 times—nearly 50 per cent. These figures indicate that tonsillar infections occurred with equal frequency in both diabetic and nondiabetics. Another noteworthy observation is, that the number of tonsillectomies in the diabetic group was much smaller than in the nondiabetic. These diabetics carried diseased tonsils into the fifth and sixth decade of life, as our physical examinations revealed, and may have suffered the ill effects of a long-continued focal infection.

TONSILLAR DISEASE AND RHEUMATIC FEVER

While the relationship between rheumatic fever and valvular disease of the heart is definitely established, it is important to realize that the relationship between tonsillar infection and rheumatic fever is not at all a settled question. Chart 1 shows that in 1530 patients with all diseases, there were 685 that gave a history of tonsillar disease. In this large tonsillar group there were 156 cases of rheumatic fever, a ratio of four tonsillar cases to one case of rheumatic fever. Fifty per cent of the rheumatic fever cases had valvular disease of the heart, which would make the ratio of tonsillar disease to valvular disease in our cases eight to one. On going over Chart 2, it will be noted that there was a history of only 125 cases of tonsillar disease with 170 cases of rheumatic fever, showing a relatively insignificant number of tonsil cases. Judging by this experience and by bedside observations in patients with rheumatic fever, it would seem that the tonsillitis of rheumatic fever should be considered as something specific for that disease and as part of the acute infectious disease itself; just as sore throat or tonsillar involvement is but a part of the clinical picture of scarlet fever, and not its cause.

THE RHEUMATIC HEART

As previously stated, valvular disease of the heart develops in more than half of the patients having rheumatic fever. Under such conditions, in the 37 diabetics who had rheumatic fever, we should have

found valvular disease of the heart in 18 or 19 cases. It is therefore highly surprising to note that in these 37 patients only one case of valvular disease was discovered. This is an unexpected finding and calls for a better understanding. Joslin noted this and says, "Rheumatic heart disease is of extraordinary rarity. In 1917 I could recall no case of diabetes under fifty years of age in which the patient complained of shortness of breath or presented the signs of an incompetent heart due to valvular disease of a definite and unquestionable rheumatic origin."[2] Later he did find one case. He says further that, "Myocardial disease in contradistinction to valvular disease is not infrequent among the diabetics."

One may deduce from these observations that when rheumatic fever attacks an individual who is a potential diabetic, or rather, one who is destined to become diabetic, such a patient's heart will be comparatively immune to endocardial involvement.

SUMMARY

The usual frequency of acute infectious diseases as they occurred in a group of 1530 patients coming under my observation is recorded. Special care was exercised in obtaining a correct history of these patients.

In the diabetics studied here, a history of rheumatic fever prior to the diabetes, was obtained with the same frequency as in nondiabetics. In 350 diabetics, we found that 37 gave a history of having had rheumatic fever at some time prior to their diabetes. In 37 nondiabetics, the expected number of cases of valvular disease of the heart would be about 18 or 19. In 37 diabetics who had suffered rheumatic fever, only one case of valvular disease of the heart was discovered.

In the light of our present knowledge, only one explanation can be offered for this extraordinary find, and that is, individuals with certain hereditary constitutional traits have tendencies to certain diseases and at the same time, immunities to other diseases or their complications.

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[2]Idem: pp. 587, 588.

A NOMENCLATURE FOR CARDIAC DIAGNOSIS*

Approved by the American Heart Association

The nomenclature as printed below represents, on the part of the committee charged with its preparation, not only much patient and painstaking labor but also, often, a generous subordination of personal preferences in the interest of harmony and unanimity. The desirability of a uniform terminology and method of formulating diagnoses is so obvious as to require no argument. It is to be hoped that the schema here presented will be deemed worthy of general adoption.—Editor.

THE widespread recognition of the need for a uniform system of terminology in cardiac diagnosis has led to the present effort to establish a nomenclature which not only will meet the requirements of heart clinics and general practitioners but will be found acceptable for use in Hospital and Insurance Classifications of Diseases and, as far as possible, in the International List of Causes of Death.

From a study of the following tables it will be seen that a complete diagnosis is intended to include not only a statement of the structural changes in the heart but also one concerning the cause of such changes, one indicating the type of disturbance of physiological function, and finally one defining the functional capacity of the heart.

Such a comprehensive diagnosis obviously requires careful consideration of every aspect of each case and is therefore essential to sound prognosis and effective treatment. Moreover the general use of such a nomenclature and such complete diagnoses should aid greatly in the accumulation of statistics sufficiently comprehensive and accurate to be of real and permanent value.

Each diagnosis, to be complete, should include one or more titles from each of the groups A, B, C, D. Groups E and F, of course, require only reference to the etiological factor.

Heart Disease

A—Etiological

1—Unknown

2—Rheumatic fever†

- a—Polyarthritis
- b—Chorea
- c—Growing pains
- d—Tonsillitis
- e—Pharyngitis
- f—Others, as purpura, erythema nodosum, etc.

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*Prepared by the Committee on Research of the American Heart Association:
Walter H. Hamburger, M.D., Chicago
Burton E. Hamilton, M.D., Boston
James B. Herrick, M.D., Chicago
Harold M. Marvin, M.D., New Haven
Hugh McCulloch, M.D., St. Louis
W. C. Munley, Captain, M.C. U.S.A.
Merrill M. Myers, M.D., Des Moines
Harold E. B. Pardee, M.D., New York
Homer F. Swift, M.D., New York
Paul D. White, M.D., Boston
John H. Wyckoff, Jr., M.D., New York.
William D. Stroud, M.D., Philadelphia, Chairman.

†In the case of each of these conditions, it should be noted, if possible, whether the etiological factor is still active or is inactive.

3—Syphilis*
4—Bacterial infection*
Specify bacterium if possible
Here should be classified subacute infective endocarditis (i.e., Streptococcus viridans), chronic endocarditis, etc.
5—Thyroid*
a—Hyperthyroidism
b—Hypothyroidism
6—Toxic
a—Bacterial toxin
b—Mineral
c—Vegetable
Specify if possible
7—Neurosis (Cardiac)
8—General systemic disease
Specify the disease
e.g., Arteriosclerosis, chronic nephritis, diabetes, emphysema, hypertension, anemia, etc.
9—Trauma
10—Congenital developmental defect

B—Anatomical

1—Undiagnosed
2—Atrophy of heart
3—Enlargement of heart
4—Hypertrophy of heart
5—Dilatation of heart
6—Ventricular preponderance
a—Right
b—Left
7—Auricular hypertrophy
8—Cardiac thrombosis
9—Cardiac infarction
10—Rupture of heart
11—Myocarditis, acute
12—Myocarditis, chronic
13—Fatty infiltration
14—Fatty degeneration
15—Endocarditis
a—Acute
b—Chronic
Include the continuous activity of a valvulitis, or papillary muscle, chorda tendineae and mural infection (usually 15 would always precede the 16 diagnosis).

*In the case of each of these conditions, it should be noted, if possible, whether the etiological factor is still active or is inactive.

16—Cardiac valvular disease
- a—Aortic insufficiency
- b—Aortic stenosis
- c—Mitral insufficiency
- d—Mitral stenosis
- e—Pulmonic insufficiency
- f—Pulmonic stenosis
- g—Tricuspid insufficiency
- h—Tricuspid stenosis

Should be thought of as active or inactive as expressed under etiology.

17—Congenital abnormality

Specify lesion if possible

18—Pericarditis, acute
- a—Serous
- b—Serofibrinous
- c—Purulent

19—Hydropericardium

20—Hemopericardium

21—Pneumopericardium

22—Adherent pericardium

23—Aortitis
- a—Without dilatation
- b—With dilatation

Aortitis (syphilis) in contradistinction to 28, which includes arteriosclerotic changes in the aorta.

24—Tumor of —

Specify part affected

25—Aneurysm of —

Specify affected vessel

26—Embolism of —

Specify affected vessel

27—Thrombosis of —

Specify affected vessel

28—Arteriosclerosis of —

Specify locality, as coronary arteries, cerebral arteries, etc.

29—Arteritis of — (except aorta)

Specify locality

30—Periarteritis of —

Specify locality

31—No circulatory disease

32—No cardiac disease

C—Physiological

1—Regular sinus rhythm

2—Ectopic rhythms (without tachycardia)

This is to be differentiated from ectopic rhythm with tachycardia of the paroxysmal type. In the ectopic rhythms without tachycardia, it is usually believed that the new rhythm has developed because of a depression of the normal pacemaker. Example: Under this heading should be classified such a rhythm as nodal rhythm.

3—Vagal arrhythmia
- a—Sinus arrhythmia
- b—Sino-auricular standstill
- c—Simple bradycardia
- d—Ventricular escape
- e—Wandering pacemaker

4—Sinus tachycardia (not paroxysmal)

5—Premature contractions
- a—Auricular
- b—Junctional
- c—Ventricular

Ventricular tachycardia (not paroxysmal)

Those which have been reported showing this arrhythmia associated with digitalis poisoning should be classified here.

- d—Unknown

6—Paroxysmal tachycardia
- a—Auricular
- b—Junctional
- c—Ventricular
- d—Unknown

7—Auricular flutter
- a—Paroxysmal
- b—Chronic

8—Auricular fibrillation
- a—Paroxysmal
- b—Chronic

9—Ventricular fibrillation

10—Auriculoventricular heart-block
- a—Partial block
  - (1)—Prolonged conduction time
  - (2)—Occasional dropped beats (irregular)
  - (3)—High grade block (2-1; 3-1; etc.)
- b—Complete block

11—Intraventricular block
- a—Partial
- b—Bundle-branch—right or left

12—Pulsus alternans

13—Valvular incompetency
- a—Mitral insufficiency
- b—Tricuspid insufficiency
- c—Pulmonic insufficiency
- d—Aortic insufficiency

14—Hypertension

15—Congestive heart failure

16—Effort syndrome

17—Anginal syndrome

D—Functional Capacity

1—Patients with organic heart disease, able to carry on ordinary physical activity without discomfort.

2—Patients with organic heart disease, unable to carry on ordinary physical activity without discomfort.
- a—Activity slightly limited
- b—Activity greatly limited

3—Patients with organic heart disease and with symptoms or signs of heart failure when at rest, unable to carry on any physical activity without discomfort.

E—Patients with Abnormal Signs or Symptoms

Not believed to be due to organic heart disease.
This diagnosis to be Possible Heart Disease, Class E.

F—Potential Disease

Patients without circulatory disease whom it is advisable to follow because of the presence or history of an etiological factor which might cause disease.
This diagnosis to be Potential Heart Disease, Class F.

Examples

Heart Disease: A.—Active syphilis; B.—Aortitis with dilatation, Aneurysm of the ascending aorta; C.—Regular sinus rhythm; D.—Class 2a; or

Heart Disease: A.—Inactive rheumatic fever (chorea); B.—Valvular disease, mitral insufficiency and mitral stenosis; C.—Auricular fibrillation; D.—Class 2b.

Department of Reviews and Abstracts

Collective Review

ETIOLOGY OF ESSENTIAL ARTERIAL HYPERTENSION*

L. H. Newburgh, M.D., and Sarah Clarkson, M.D.
Ann Arbor, Mich.

I

At the outset it is desirable to remind the reader that arterial hypertension is a disturbance of function. Since the pressure of the blood is produced by a force pump (the heart) driving against a peripheral resistance, it is evident that no degree of morphological change can cause elevation in blood pressure in a nonliving organism. The maximum pumping effect of the heart alone is not sufficient to maintain a pressure which will permit glandular organs, such as the kidney, to function. Likewise, normal pressure cannot be maintained against maximal peripheral resistance in the presence of an incapable heart muscle. The peripheral resistance is brought about chiefly by varying degrees of contraction of the arterioles. Their caliber is controlled by the vasomotor nervous system which has a center in the medulla and subsidiary centers in the spinal cord. Destruction of the medullary center or section of the main path of this nervous system (in the rabbit) quickly causes a fall of carotid blood pressure to about 40 mm. of mercury. On the other hand, stimulation of this vasomotor system is followed by great increases in pressure.

As yet the physiologists have not discovered the mechanism which governs the activity of the vasomotor apparatus, but it is known that blood pressure may be raised to an abnormal height in a number of ways. Experimentally, electrical stimulation of the central end of sensory nerves produces a very rapid and large rise in blood pressure. The hypertensive effect of injected adrenalin is common knowledge. Pain in a human being generally has an elevating effect on blood pressure. On the other hand there is no close correspondence between the level of blood pressure and the metabolic rate, or the temperature. For, whereas in Graves' disease, the blood pressure often is slightly above the normal (5 or 10 per cent), the metabolic rate in the same individual may be increased 50 to 75 per cent. In fevers, the blood pressure tends to be too low rather than too high. As already has

*From the Department of Internal Medicine, Medical School, University of Michigan.

been stated, the blood pressure is the resultant of two forces—the propulsion of the blood by the heart, and the resistance to its outflow caused by the contraction of the arterioles. From a hypothetic point of view either one of these forces acting alone should be able to cause an abnormal elevation of the blood pressure. But it already has been pointed out that the heart acting alone, cannot maintain a normal pressure, even though the organism is dying because of the too low pressure and must need the greatest amount of blood which the heart is capable of pumping. On this basis it is a fair assumption that hypertension, or at all events continued hypertension, never is attributable to overaction of the heart alone. In fact the heart is provided with a mechanism for protecting itself against hypertension. We refer to the depressor nerve which joins the heart to the vasomotor center in the medulla and which has an inhibitory quality. When this nerve is stimulated the blood pressure falls; and Carl Ludwig, who discovered the depressor nerve, pointed out that this mechanism provided the heart with a means of lowering the pressure against which it must empty its chambers, as a protection against fatigue. On the other hand evidence already has been cited to show that hypertension easily is brought about by augmented activity of the vasomotor apparatus through its ability to increase the resistance to outflow. Accordingly, in the present state of our knowledge we may regard increase in peripheral resistance as the immediate cause of hypertension. One can picture such disturbances as occurring in the following ways.

The caliber of the small arteries throughout the body might be so decreased by thickening of the vessel wall that even though in a state of complete relaxation the path for the blood from the larger vessels to the parenchyma of the organs would be too small to furnish them with sufficient nutrition if the flow through these narrowed vessels were not continuously increased by a constant elevation of blood pressure maintained by more work on the part of the heart. The type of arteriosclerosis described by Gull and Sutton as arteriocapillary fibrosis presents an actual diseased state that corresponds well to the theoretic requirements just set down. If, however, what appears to be the present trend of classification be adhered to, arteriocapillary fibrosis may not be included as a cause of essential arterial hypertension, since the term essential is used in the sense of idiopathic, i.e., of unknown origin, or at least without any morphological basis as yet discovered.

This leaves us with those forms of hypertension which are supposed to be caused by the continuous contraction of the small blood vessels brought about by the direct action of substances on the muscles of their walls, or by substances acting upon the vasomotor center. These substances are thought of either as an excess of what is normal, or as the

presence of abnormal bodies possessed of the quality which causes overaction of the vasomotor nervous system.

Finally it seems necessary to include the idea of excessive stimulation of the sensory nerves by disease, and by certain psychic states which affect the vasomotor center through direct nervous connections, or through the intervention of endocrine glands.

II

Possible causes of essential arterial hypertension may be grouped under three headings: chemical, neurogenical, and anatomical.

A. CHEMICAL

1. *Excess of Normal Substances.*—The literature contains several attempts to show that the caliber of the peripheral vessels is regulated by a chemical hormone, and Schur and Wiesel[1] some years ago believed that they had brought forward proof that adrenalin was the material in question. Their further work convinced them, at the time, that hypertension was caused by an increased secretion of adrenalin. Subsequent studies have thrown great doubt on both hypotheses and especially on the latter. The more recent methods devised to determine the amount of adrenalin in the blood do not support the idea that clinical hypertension is caused by hyperadrenalinemia.

In this same field the careful studies of Major[2] and his associates are of great interest. He has pointed out that "The probable presence in the urine of some substance capable of exercising a profound influence on blood pressure has been much discussed. It is evident that any product of metabolism able to produce a striking rise in the blood pressure could, if not excreted properly, induce very marked changes. It is suggested that such a substance may be responsible for the high blood pressure observed in essential hypertension, in chronic nephritis, and in all allied conditions such as eclampsia. The effects of nonprotein nitrogen substances upon blood pressure were studied. While creatinine and creatine have appeared inert, certain derivatives and closely allied substances have shown marked pressor effects. The result obtained with methyl guanidine was striking." Major, et al., have shown that the intravenous injection of 0.1 gm. per kilo of body weight of methyl guanidine compounds produces a well marked and prolonged elevation of blood pressure in dogs. To quote further, "Just as striking as the prompt rise was the persistence of a high blood pressure, the second maximum being maintained until shortly before death. The powerful vaso-constrictor effect of methyl guanidine was considered as the most probable cause of the marked hypertension. Several records of the effect of methyl guanidine upon the blood pressure in man were obtained. The substance was injected intravenously in doses varying from 0.045 gm. to 0.08 gm. per kilo. No

unpleasant symptoms aside from a sense of slight fullness in the head and a slight pounding of the heart were noted. The systolic and diastolic blood pressures were promptly elevated and these elevations persisted for one hour. The systolic blood pressure was elevated in these observations, from 30 to 50 mm. and the diastolic pressure from 15 to 20 mm. The ability of methyl guanidine, a product of metabolism, to produce a marked and sustained rise of blood pressure is of considerable interest. It seems possible that this substance if produced in excess in the body, or if not excreted by the kidneys, might cause a rise of blood pressure."

Before the hypothesis that hypertension is caused by methyl guanidine compounds is accepted it must be shown that either (a) these substances are being produced in excess; or (b) that they are being retained within the body, in pertinent cases. The technic employed in the quantitative determination of methyl guanidine is fraught with great difficulties so that caution must be used in accepting the data. Howard and Rabinowitch,[3] following Major, have studied this problem in the human subject. They found marked fluctuations in the amount of methyl guanidine excreted daily. The average twenty-four-hour excretion of ten normal subjects was 119 mg. Thirteen cases of marked arterial hypertension were found to have a daily average output of the guanidines, of 105 mg. The differences here are too small to be convincing, even though it is true that the highest pressures were associated with the lowest urinary guanidine values. In one patient daily observations were made over a period of two weeks. There was a marked fall in both the systolic and diastolic pressures, and accompanying this, a great increase in the daily excretion of dimethyl guanidine. These data support Major's hypothesis but must not be accepted as final proof.

One cannot avoid asking why total nephrectomy is not followed by a steadily rising blood pressure if the latter be caused by retention of methyl guanidine. It is a fact, however, that the blood pressure is not importantly altered by the removal of the kidneys.

Nuzum and Sansum[4] in two recent publications have recorded abnormally high blood pressures in rabbits that have been on diets excessive in their protein content. They attribute the pressor effect to a shift in the acid-base equilibrium toward the acid side. Anderson[5], however, claims that a high protein diet has no effect on the blood pressure of the rabbit. The excessive use of protein by the human subject has been considered repeatedly to be the cause of arterial hypertension but no one as yet has succeeded in demonstrating this clearly to be the case. It is true that the ingestion of large amounts of protein for short periods of time does not affect the blood pressure definitely. There are no studies of this sort dealing with time periods of six months or a year. At present we are not in a position to attribute es-

sential arterial hypertension to excessive ingestion of protein, nor, on the other hand, has it been shown that there may not be a relationship between the two.

2. *Abnormal Bodies.*—From time to time observers have suggested that pressor agents were formed by bacterial activity in the gut and that the absorption of these substances caused hypertension. But to date, no one has demonstrated the presence of these substances in the blood of the living animal.

B. NEUROGENICAL

1. *Periphery.*—It is well known that pain will cause marked elevation of the blood pressure, and it is easy to include in this category the thought that all unpleasant sensations due to the physical environment may have the same effect in a cumulative way. But the recorded responses of blood pressures to pain always have been brief. We are unaware of any record of persistent hypertension caused by pain in the narrow meaning. We, however, have had the opportunity of observing a patient whose blood pressure was 300 mm. and over, systolic, whenever measured, for more than a week during which time she was suffering from renal colic which caused continuous pain. The removal of the stone was followed by a dramatic and persistent fall of the blood pressure to normal. On the other hand, it is true that the subjects of painful disease do not exhibit any important degree of prolonged hypertension, in the sense of a chronic disturbance.

2. *Psychic.*—Fear causes elevation of blood pressure as has been demonstrated by Cannon[6] in the experimental animal. Fear is a primitive sensation which in us often takes the form of anxiety, apprehension, and dread of adverse criticism. Do these psychic pains cause essential arterial hypertension? This question which is in the minds of physicians cannot be answered in any simple way. Moschcowitz[7] has crystallized a view held by many shrewd physicians. He says, "I have found that the greatest proportion of patients afflicted with hypertension conforms to a certain type, which may be described as both physical and psychic. The patients are overweight and sometimes even obese. The neck is short, the muscles are soft, their bodily movements are sluggish, their carriage and walk are ungraceful and they lack the spring and elan of the former athlete. Psychically, these people are tense; they pursue their vocation with tremendous seriousness and worry over trivialities. In consequence they are irritable. Phlegm and hypertension are, in my experience, antagonistic. Furthermore, these individuals have narrow intellectual horizons. Their interest in anything outside of their business is desultory. They have no hobbies. In their leisure hours they play cards, sit in the theatre, read the newspapers or rehearse the business activities of

the day. Their sleep is not restful and they 'do not believe in vacations.' Who cannot recognize in this picture the individual that has been termed the tired business man, whose tastes have activated a large portion of the so-called modern drama? He eats well, drinks alcohol to obtain the stimulation that his mental faculties do not afford, and his most violent daily exercise is his walk to and from the conveyance that takes him to his business. As a rule these people are 'successful' and they may be said to die of 'success.' His most conspicuous mental incapacity is his inability to play."

We know too little about constitutional types as yet to deal conclusively with this question. A relationship between personality and blood pressure no doubt exists; but our feeling is that we should regard constitutional causes of disease with suspicion rather than allow ourselves to be carried away by the conviction that the problem of essential hypertension largely is settled by identifying it with types.

C. LOCAL

In refreshing contrast to the difficult field of neurogenic causes is the simple and direct information recently published by Bordley and Baker[8]. They point out that Anrep and Starling have shown that a reduction in the amount of blood reaching the brain brings about a compensatory rise in the systemic blood pressure. With this in mind they have investigated twenty-four cases of generalized arteriosclerosis in fourteen of which there was arterial hypertension and in ten a normal or low blood pressure. They found arteriolosclerosis in the medulla in every case with a history of hypertension, whereas in the subjects with normal pressure the arterioles in the medullary region were unaltered. They conclude that there is at least an association between persistent hypertension and arteriolosclerosis of the vessels supplying the medulla, and suggest the possibility that the inadequate medullary blood supply brought about by the narrowing of the vessels is corrected by a systemic increase in blood pressure. If this should be proved to be a fact, we must regard this particular form of hypertension at least as beneficent.

Paullin[9] recently has made a significant contribution to our understanding of hypertension. His study deals with sixty-six cases followed for at least five years and which were about equally divided between the two sexes. The mortality for the men was 48.7 per cent whereas for the women it was only 9.2 per cent. Furthermore half of the cases in women occurred in association with the menopause—fifteen of these sixteen patients still were living at the time of publication, and in five of these (or one-third) the blood pressure again has become normal. It would appear from this study that essential hypertension may be differently caused depending upon sex, and that

the etiological factors common in the male group are of much less importance among the women.

CONCLUSION

Blood pressure is the resultant of two forces. Of these, increases in the peripheral resistance alone are responsible for essential arterial hypertension. The great variety of factors which may cause such increases have been discussed but it is not possible at present to determine which one of these hypothetical factors is responsible for the hypertension in the individual case. As the investigation of the etiology of essential arterial hypertension has advanced, an increasing number of possible causes of this condition has been found. The progress in this field indicates that the term essential arterial hypertension is a generic one and that gradually a number of types, each with its own etiology, will be recognized.

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[7]Moschcowitz, Eli: Hypertension, Its Significance, Relation to Arteriosclerosis and Nephritis, and Etiology, Am. Jour. Med. Sc., 1919, clviii, 668.

[8]Bordley and Baker: A Consideration of Arteriosclerosis of the Cerebral Vessels and the Pathogenesis of Hypertension, Johns Hopkins Hosp. Bull., 1926, xxxviii, 320.

[9]Paullin, J. E.: Ultimate Results of Essential Hypertension, Jour. Am. Med. Assn., 1926, lxxxvii, 925.

NOTE.—A full discussion of all the earlier work and views on essential hypertension (hyperpiesia) and of the relations between arterial hypertension and disease of the arteries and kidneys will be found in Sir Clifford Allbutt's classic treatise on "Diseases of the Arteries," London, 1915.

Selected Abstracts

Kilgore, Eugene S.: Angina Pectoris and Pseudo-Angina; A Clinical Study with Special Reference to Lancinating Pain. Jour. Am. Med. Assn., 1926, lxxxvii, 455.

The material for this study is summarized from the records of 253 patients complaining of pain in the region of the heart and in two instances pain elsewhere which was thought to be related to the heart or aorta. The fact that most of these patients were young men makes the series unsuitable for study of age and sex distribution of pericordial pain for the population at large. The 36 cases diagnosed angina show the usual predilection for males and for middle and advanced age.

One hundred "normals" were normal in the sense of lacking hypertension or demonstrable circulatory distress. The remaining 153 cases showed definite structural change in the circulation. The diagnoses of angina were made according to the usual rules of differential diagnosis.

Although many descriptive terms were used by these patients to describe their painful sensations, it was found that most of them were readily classified into three main types which may be termed (1) lancinating; (2) dull; (3) compression pain. What is here designated as "dull pain" is the assortment remaining after taking out those with distinctive lancinating or compression qualities. A study of the frequency of these three terms shows that lancinating pain predominates and with few exceptions is in the pericordial region. Dull pain comes next in frequency and is also mostly pericordial but not infrequently substernal and compression pain, while distinctly less frequent in general, predominates in the central chest location and is about equally common in the precordia. The small angina group is conspicuous for its paucity of lancinating pain. Of these 36 cases which the author believed to be genuine angina, only 4 patients described lancinating pain and in only one of these was it unassociated with other pain suggesting the compression qualities.

Rappaport, B. Z.: Primary Acute Aortitis. Arch. Path. and Lab. Med., 1926, ii, 653.

A case of primary acute aortitis due to Streptococcus viridans is reported with no aortic endocarditis present. A syphilitic aortitis was associated with the acute aortitis.

From a study of the literature and of this case, the author points out that previous pathological changes in the vessel, whether atherosclerotic or syphilitic, predispose to acute infection of its wall either through direct implantation on its surface or through the vasa vasorum.

The presence of a concomitant acute inflammation does not influence the histological picture of syphilitic aortitis. The presence of polymorphonuclear leucocytes in a lesion even with definite evidence of syphilitic changes should lead to a careful and thorough search, not only for spirochetes, but also for pyogenic organisms as a possible cause for the acute reaction.

Musser, John H., and Herrmann, George R.: Chronic Pericarditis. Jour. Am. Med. Assn., 1926, lxxxvii, 459.

Acute or chronic pericarditis is one of the pathological lesions which the clinician rarely diagnoses. In explanation the authors point out (1) the pathognomonic signs are looked for and if not observed the case is dismissed as one without pericardial involvement. (2) The dogmatic statements of textbooks are that such and such signs are present, when actually they are present only occasionally or are entirely absent. (3) The vividness of the symptoms of existing cardio-circulatory failure conceal the less obvious signs of pericardial disease which may be responsible for the circulatory embarrassment of the patient; also, probably of greater importance than these, there is a well known fact that pericarditis rarely if ever exists as a primary disorder.

In an endeavor to discover why pericarditis is generally overlooked, and to see whether they could not find some useful data that might aid in the diagnosis of this disease, the authors have reviewed all necropsy and clinical records of proved cases of pericardial involvement at the Charity Hospital in the five years 1921 to 1925. Among these 1720 necropsies, record is made of 305 cases that show a pericardial involvement. Of these 39 presented pericardial lesions which the pathologist believed were contributory causes of death. Pericarditis either acute or chronic was found in 11.9 per cent of the bodies at necropsy. In addition to the 39 cases there were 29 cases in which, from the records of necropsy and the history of the patient, it was believed that the pericardial condition also played a part in hastening death.

The symptomatology of chronic pericarditis seems to belong entirely in the silent group as there are no symptoms until the development of cardiac failure that might suggest the presence of pericardial disease. It is necessary to depend almost entirely on the examination of the patient. As to the possibility of an adherent

pericardium being present without symptoms or signs, we may assume such to be the case (1) in the young person with a chronic valvular disorder associated with considerable hypertrophy of the heart; (2) when there is valvular disease of the heart which fails to respond to digitalis; (3) in those patients whom the physician had the opportunity of seeing when an acute pericardial lesion was demonstrable.

Campbell, J. M. H., and Poulton, E. P.: The Effect of Respiratory Exchange in Chronic Bronchitis. Quart. Jour. Med., 1926, lxxvii, 49.

The exercise tests shown were similar to those described in the previous paper.[1] A closed circuit apparatus was employed and the metabolism was taken before exercise.

The writers used the figure 0.157 c.c. per step per kilo, as being the oxygen equivalent of the work done and they based their calculation of the percentage efficiency of the individual on the amount of oxygen which was used beyond this figure.

It was found that on exercise the breathless subject required more oxygen than the healthy; this difference was slight and was found to be due to the delayed return to the resting level rather than to the increased consumption during exercise. The percentage efficiency is thus decreased but this factor was inadequate in itself to account for the breathlessness.

The carbon dioxide in those cases was more strikingly altered than the oxygen absorption. The output showed a slow rise during exercise, and a gradual fall to the resting condition afterwards.

The Respiration Quotient was low and it was thought that either the carbon dioxide was slowly excreted sometime after the experiment was completed, or that some qualitative change took place in the metabolism. An attempt was made to correlate the breathlessness of these patients with the changes in their pulmonary ventilation. In all of these subjects shallow rapid breathing was present, and this is held to account for the carbon dioxide deficiency in some cases, depending on the type of pulmonary ventilation.

One group less breathless showed no increased ventilation, a normal oxygen consumption and a delayed carbon dioxide excretion. Here the slight breathlessness stimulated the respiratory center and ventilation was increased with a fair gaseous exchange. The second group included very breathless patients where pulmonary ventilation was deficient and where oxygen consumption and carbon dioxide excretion were delayed. It is probable that in this second group pulmonary obstruction led to exhaustion of the respiratory center with consequent underventilation.

[1]Campbell, J. M. H., and Poulton, E. P.: Effect of Exercise on the Pulmonary Ventilation and Rate and Depth and Breathing in Chronic Bronchitis, Quart. Jour. Med., 1926, lxxvii, 27.

It is suggested that in bronchitis the respiratory center responds by rapid shallow breathing. If the bronchitis is followed by emphysema, the breathing remains shallow; if asthma predominates, breathing is deepened and gaseous exchange is incompletely carried out.

Campbell, J. M. H., and Poulton, E. P.: Effect of Exercise on the Pulmonary Ventilation and Rate and Depth of Breathing in Chronic Bronchitis. Quart. Jour. Med., 1926, lxxvii, 27.

This study was based upon observations on the exercise reaction of patients with chronic bronchitis. The normal individual reacts to exercise by increasing first, the depth of breathing and if exercise is continued the rate of respiration also increases.

The test used consisted in stepping on and off a thirteen inch step; and the duration and rate of this exercise varied somewhat with the cases studied. At rest the patients with bronchitis had rapid shallow respirations, and it was found that with exercise the ventilation was increased about as much as in health, although compared with the resting respirations of these cases the increase was relatively less. The rate of respiration rose and the depth was decreased.

In one patient with myocardial disease and with pulmonary signs, the ventilation, which was normal before exercise, increased afterwards; the breathing became deeper but not more rapid, and after ten minutes it had not returned to normal.

The respiration rate in chronic bronchitis was shown to vary somewhat with the duration of the disease being usually higher in those cases of longer standing. In all the cases a delay in return to the preexercise level was seen. The vital capacity of these patients was studied. Very little relation was shown between the ability for exercise and the capacity of the chest. The values for the patients with bronchitis were low; for the cardiac normal in spite of severe dyspnea in the latter.

The pulse rate of these subjects was also observed.

It was found that in some the rate increased with exercise, in others no change took place. This pulse reaction bore no relation to the respiratory signs which occurred.

Morris, Roger S.: The Clinical Recognition of Pulsus Alternans. Jour. Am. Med. Assn., 1926, lxxxvii, 463.

In the eight months preceding this report, 19 cases of constant alternation of the pulse had been observed by the author either in the hospital wards or in consultation practice. Of these, 7 had had alternation in the intensity or pitch of the heart sounds or murmurs. Auscultation has given more valuable information than palpation over the brachial artery, although with high pulse pressures palpation may

give striking findings. Eight of the author's 19 patients have died within nine months and 5 more were known to be in a critical condition.

The blood pressure findings which are characteristic of pulsus alternans may also be found in patients in whom the respiratory rate is one-half of the pulse rate and also in those with bigeminal rhythm in whom the premature contractions are late and can be recognized only by graphic methods.

Bramwell, J. Crighton, and Hickson, Sylvia K.: The Relation of the Pulse Form to Sound Production in the Arteries, Part II, Phonoarteriograms. Heart, 1926, xiii, 129.

In a second paper dealing with the demonstration of the Korotkow sounds an optical method was described. The recording apparatus consisted of a pneumatic armlet, a receiver for the brachial arteries connected by pressure tubing to a Frank's segment capsule with attached mirror. Two points in the apparatus were stressed. The open side tube which put the pressure tubing in communication with the outside air and the delicate membrane of fresh rubber used in the capsule.

It was shown that oscillations were produced which were constant and which were due to the vibration of the air in the pressure tubing caused by the vibration of the front of the pulse wave in the artery distal to the armlet. In addition waves preceding the initial deflection were seen during the early stages of decompression. They were analogous to the preanacrotic waves of the sphygmogram and their disappearance synchronized with the occurrence of the Korotkow sounds. These last waves are not considered instrumental in origin but due to the actual movements of the arterial wall.

Brooks, Barney: Ligation of the Aorta. Jour. Am. Med. Assn., 1926, lxxxvii, 722.

A negro with classical signs and symptoms of a very large abdominal aneurysm revealed the following significant facts on examination: (1) the cause of the aneurysm was syphilis; (2) the heart was essentially normal; (3) there was a complete absence of pulsation in the left femoral artery and a very feeble pulsation in the right femoral artery; (4) the left lower extremity was considerably swollen and also was distinctly cooler than the right. The general condition of the patient was such as to indicate that he was a good operative risk.

At operation, when an attempt was made to enucleate the large aneurysmal tumor, a rent was made in the mass and it became necessary to ligate the aorta. Knowing that the application of a ligature in the usual way would, in all probability, result fatally from its

cutting through, the author sought to prevent this by the application of a ligature made of a piece of fascia lata excised from the thigh. The strip of fascia lata, cut approximately 0.5 cm. in width, was passed around the artery twice just distally to the origin of the inferior mesenteric artery. The fascia lata was tied tightly enough to stop all pulsation distally. A braided silk ligature was then tied tightly about the aorta just proximal to the aneurysmal tumor. There was a space of approximately 1 cm. between the two ligatures. The abdominal cavity was closed. The patient eventually recovered and was discharged from the hospital. He was seen from time to time and it was noted that the aneurysmal tumor gradually grew smaller and at no time showed any evidence of pulsation. Two months later he was readmitted to the hospital because of abdominal pain caused by an intestinal obstruction at the site of the previous operation. He died and a postmortem examination revealed that the aneurysmal tumor had diminished to a hard mass about the size of a small orange which was firmly fixed to the vertebral column. The sac was completely obliterated. The aorta was carefully opened at the site of ligation. It was completely obstructed, the occlusion of the aorta being due to a well organized thrombus at the site of ligation. The fascia lata was easily identified by the knot of fascia through which a black silk ligature had been placed at the time of operation. The knot remained remarkably well preserved. The fascia encircling the aorta, however, had become so confluent with the fibrous tissue about that it could not be traced. The braided silk ligature was found wholly in the organized thrombus in the artery. Both external iliac arteries and hypergastric arteries were patent. The right common iliac vein appeared normal. The left common iliac vein had been completely obliterated in the aneurysmal tumor.

Kaiser, A. B.: Tonsillectomy in Children. Jour. Am. Med. Assn., 1926, lxxxvii, 1012.

The present paper is a study of a group of 8000 children between the ages of five and ten years who were admitted to a clinic for tonsillectomy. The author describes the indications for tonsillectomy in children based on end-results in this group. The children have been observed for a period of five years.

The significant statement in relation to heart disease is made that much may be hoped from tonsillectomy in reducing the incidence of the so-called rheumatic syndrome. Chorea had existed in 7 of the children not operated upon in the control group and in 8 of the group operated upon. Six of the 8 children developed the symptoms since removal of the tonsils. A history of rheumatic pains was obtained in equal numbers in the two groups. Cardiac disease was

based on the physical examination. In the control group 52 children presented evidence of cardiac disease. In the group operated upon there were 44 cases, of which number 13 had developed since the operation. With the decided lower incidence of sore throat in the group operated upon it is hoped that cardiac disease also will be less likely to develop in these children as the years go on.

Sigler, Louis H.: Periodic Variability of Some Forms of Arterial Hypertension. Amer. Jour. Med. Sci., 1926, clxxii, 543.

Five cases are described with periodic variations in blood pressure readings ranging from marked hypertension to subnormal. The pathological changes in the circulatory and renal structures as shown by clinical manifestations could not account for these variations nor could such variations be accounted for by any change in the mode of living of the patients. It would seem that variations of this kind, as well as hypertension in general, will depend upon some structural or functional abnormality in the vegetative nervous system, exhibiting itself in the form of vasomotor pressor effects.

McLester, James S.: The Causes and Treatment of High Blood Pressure. Amer. Jour. Med. Sci., 1926, clxxii, 643.

The article is a brief concise statement of the most recent conceptions of the causes of high blood pressure. He concludes that today we must regard high blood pressure merely as a disease complex often inherited in which hypertension is the salient clinical feature. The exact pathogenesis is unknown, but in its later stages it is accompanied by disease of the kidneys, myocardium and arteries. For simplicity's sake, the following classification may be adopted: (1) early hypertension; (2) chronic hypertension without nephritis, so-called essential hypertension; (3) chronic hypertension with nephritis; (4) fulminant hypertension.

The treatment of arterial hypertension, though sometimes fruitless, will often accomplish much. For patients with long standing hypertension and pronounced change in the arteries, heart and kidneys, one can do little else than temporarize and postpone the inevitable, but for those whose illness is of shorter duration and who present less obvious organic changes, one can accomplish a great deal. The author states that the manner of living for these patients counts most of all. Diet is of great importance. The correction of obesity is often an essential factor in the treatment. The removal of all foci of infection is necessary.

The hazards to be met in this disease are threefold: renal, cardiac and arterial. When disaster threatens, treatment must be addressed to the source from which the danger comes.

White, Paul D.: The Prognosis of Angina Pectoris and of Coronary Thrombosis. Jour. Am. Med. Assn., 1926, lxxxvii, 1525.

The author has studied a group of 200 consecutive cases of angina pectoris seen in private practice in the last five years with a view to determine whether or not there may be data of value on which to build a prognosis in angina pectoris. An additional reason for this study was to determine what the expectation of life in angina pectoris may be, so that there will be some data for comparison with that eventually collected from a series of cases treated by cervical sympathectomy or other special therapeutic measures. The author also includes a group of 62 consecutive cases of coronary thrombosis for comparison. Sixty-six of these 200 patients have died and 134 are alive, according to recent reports. The author presents a comparative analysis of the various phenomena associated with this group of patients in relation to prognosis.

The series of 62 cases of coronary thrombosis included 32 of the 45 patients with angina pectoris who had coronary thrombosis also. Of the total series 32 have died, all of cardiac deaths except one who had gastric carcinoma.

It may be said that hypertension, coronary thrombosis, syphilis, evident arteriosclerosis, poor heart sounds, abnormal T-waves in the electrocardiogram and especially cardiac enlargement are more often found in patients with angina pectoris who succumb within a few years than in those who survive. Sex, occupation and direction of radiation of anginal pain do not affect the prognosis. Patients with coronary thrombosis survive for years in good or in fair condition. The average duration of life after the attack in this group is close to two years. Hypertension, evident sclerosis and syphilis alter the prognosis hardly at all. The heart is enlarged in the majority of patients in both living and dead groups.

Boas, Ernst P., and Fineberg, Meyer H.: Hypertension and Its Relationship to Mitral Stenosis and Aortic Insufficiency. Amer. Jour. Med. Sci., 1926, clxxii, 648.

During the four years covered by this study, 135 patients were discharged from or died in the hospital with the diagnosis of mitral stenosis uncomplicated by aortic disease. Of these, 39 or 29 per cent had hypertension. Hypertension was much more common in women than in men with mitral stenosis and in the group of patients with both conditions 69 per cent were women except in 5 cases in which hypertension associated with mitral stenosis occurred in patients over forty years of age. Of the 68 cases under forty years of age 7 per cent exhibited hypertension, while of 67 cases over forty years of age 55 per cent had an elevated blood pressure. During the same period

of the study 60 cases of uncomplicated aortic insufficiency were observed. Sixteen, or 27 per cent, of these had hypertension. No comparison of the sexes is possible for in the whole series there were only 13 women. Hypertension occurred only in patients forty years of age or older; it was relatively uncommon in the rheumatic or syphilitic group but one-half of the patients with arteriosclerotic aortic insufficiency had an associated hypertension. Only 8 per cent of patients with both mitral stenosis and aortic stenosis had an accompanying hypertension.

The authors conclude that hypertension often of considerable degree is a common complication of chronic heart disease. It is more frequent in patients over forty years of age. In chronic heart disease it is found more often in patients with mitral stenosis occurring in fully 5 per cent of those over forty years of age. It is suggested that granular kidneys found at autopsy in these patients are caused by arteriosclerosis consequent on a long standing hypertension.

Edwards, D. J., and Page, Irvine H.: The Effects of Parathyroid Extract on the Heart and Circulation. Amer. Jour. Physiol., 1926, lxxviii, 235.

Subcutaneous injections of parathyroid hormone were given to healthy dogs at intervals of about two to four hours. After several doses of the extract had been given, blood samples showed calcium values between 15 and 22 mg. per cent. Measurements of the blood viscosity were made in some experiments and showed an increase in this property. The animals showed fine muscular tremor very early in the course of the injections. Later, however, there was always some depression and in a few instances an unmistakable atonia.

The heart rate increases slightly during the early stages of the experiment. This is followed by a slowing and usually a marked arrhythmia. Premature beats and a shift of the pacemaker function frequently appear during this stage. The electrocardiographic records do not reveal any constant changes.

Lewis, Thomas, and Zotterman, Y.: Physical Reaction of the Skin to Injury. Annular Edema of the Skin in a Case of Infective Endocarditis. Heart, 1926, xiii, 193.

The authors studied the annular skin rash occurring in a case of subacute bacterial endocarditis and showed the mechanism whereby annular eruptions of toxic origin were produced.

The lesions studied began as papules which became umbilicated and later spread out coalescing into rings of edema with central areas of pigmented skin. Where two such rings met growth in that direction ceased. New papules never appeared over an old lesion but either on

fresh skin or on the outer edge of a wave of edema. When in the latter position no umbilication occurred and the lesion was not characteristic. These points emphasize the inability of the injured vessels to react further to the toxin. It was further found that an injection of histamine did not produce the characteristic wheal when injected into the central pigmented area and only produced a modified reaction when injected into the margin. Adrenalin injected in the same manner gave normal results. It was found too that raising the venous pressure resulted in petechiae in the affected areas, while only slight ecchymosis was seen in the skin elsewhere.

From these observations the authors conclude that toxins act on the skin by producing vasodilatation, increased permeability and fragility. A certain refractoriness of the vessels is present and it is this irresponsiveness that governs the spread and annular configuration of the lesion.

Lewis, Thomas: Vascular Reaction of the Skin to Injury. An Irresponsive Condition of the Vessels with Special Reference to the Pathology of Telangiectases and Allied Conditions. Heart, 1926, xiii, 153.

Experimental refractoriness of the cutaneous vessels was produced in various ways. The method previously used by Grant and Lewis was cited where histamine was used. When histamine in 1:3000 solution was injected intradermally after occlusion of the circulation in the limb, no wheal resulted even after the circulation was restored. The same lack of response occurred in a case of urticaria when the arm was similarly treated then stroked. The area was refractory not only to stroking but to histamine and morphine. The effect of freezing the skin was also studied. It was shown that the wheal produced was similar to that of histamine and that a refractoriness existed in the frozen skin to further freezing or to wheal formation. Burns from heat and from ultraviolet rays were shown to produce analogous results.

The mechanism underlying these reactions is supposed to consist in a release of histamine-like substances in the skin and these act only on the permeability of the vessels though the exact nature of the refractoriness is undecided. The vasodilatation which accompanies the whealing is due to a local nervous reflex and does not appreciably change with repeated injections.

Adrenalin injected into any of these areas does not produce the classical blanching. This shows the inability of the skin vessels to contract after the liberation of this histamine-like substance.

Certain pathological conditions bearing on this skin refractoriness are discussed. Erythema ab igne, telangiectases due to injury or of

unknown origin, and congenital naevi were studied. In all of these there is a defective vasoconstriction to adrenalin and a failure of response to histamine. This suggests that the vessels in those conditions resemble vessels after injury, and in telangiectases a traumatic origin is most probable.

Salant, William, and Nadler, J. Ernest: The Relation Between Cardiac Reactions to Drugs and the C_H of the Blood. Amer. Jour. Physiol., 1926, lxxviii, 308.

The authors studied the effect of caffeine injections into different animals shortly after the introduction of effective doses of alkali into the circulation.

The disturbance of acid base equilibrium of the blood modifies the effect of caffeine on the heart in different animals. When the P_H of the blood is increased above normality, caffeine stimulates the heart but when the alkalinity is increased beyond these limits caffeine produces depression. Caffeine also depresses the heart when the P_H of the blood is below normal. Caffeine depresses the auricles more than the ventricles both when alkalinity is excessive as well as when the C_H is increased. The authors discuss the mechanism of the action of caffeine under the conditions described. It is suggested that acid sensitized the inhibitory and alkali the augmentor mechanism of the heart and that caffeine acted on the mechanism which was sensitized most.

Bazett, H. C., and Sands, Jane: An Experimental Study of Chronic Aortic Regurgitation in Dogs. Jour. Clin. Inves., 1926, iii, 65.

The authors attempted to produce injury to the aortic cusps of dogs under conditions where there was no infection and where myocarditis could be excluded. The animals were kept for periods varying from two to eleven months after operation. Under light anesthesia a button-shaped metallic stillete was introduced through the carotid artery into the aorta and injury to the aortic cusps produced by pushing through the cusps. One, two and three cusps were injured in this manner. The animals were studied by x-ray, electrocardiograms and postmortem findings.

Chronic aortic regurgitation produced in this manner is followed immediately by a diminution in the size of the heart shadow, though later this shows a gradual increase. The pulse rate is raised immediately as a result of the lesion, but the change is rarely sufficient to account completely for the diminution observed in the heart shadow. The pulse rate never returns to its previous low level. The increase in heart size commences probably within the first week and continues rapidly for a period of one to four months. From four months up to

one year the change is still detectable but is quite gradual. The hypertrophy of the left ventricle is associated with an increase in the cavity which is often more obvious than the thickening of the wall. Hypertrophy does not depend on infection. The total lack of symptoms in most cases is astonishing and the ability to do work is often very great, though probably lessened as a result of the operation.

Tappan, V., and Torrey, E. H.: Studies on the Cardiac Output of the Dog. Amer. Jour. Physiol., 1926, lxxviii, 376.

Changes in the pulse rate of dogs under deep morphine anesthesia brought about through vagus influence exert no apparent effect upon the output of the heart. On the other hand, under urethane and pantopon anesthesia, the output of the heart may be markedly changed by similar changes in pulse rate. The present report is limited to observations upon the minute volume as influenced by changes in pulse rate in 21 dogs. The output of the heart was determined by the Fick principle.

Tulgan, Joseph: Further Evidence on the Physiological Maximum of the Heart. Amer. Jour. Physiol., 1926, lxxviii, 1.

The author concludes from his study that the denervated heart cannot be considered to respond to certain changes in the animal in the same way as the heart which is still innervated by the sympathetics but with the vagi sectioned. Further evidence is presented to show that the physiological maximum rate of the heart is obtained only after bilateral vagotomy. There is no evidence at present that the denervated heart departs from the law of the physiological maximum rate.

Rabinowitch, I. M., and Bazin, Eleanor V.: The Output of the Heart per Beat in Hyperthyroidism. Arch. Int. Med., xxxviii, 566.

The authors studied the measurement of the output of the heart beat by direct determination of the oxygen content of arterial and venous blood in a series of cases of hyperthyroidism. Twenty cases were selected whose basal metabolic rates ranged between +15 and +36 per cent. All cases belonged to the exophthalmic goiter, or toxic hyperplastic group. In no case was there evidence suggestive of heart failure other than tachycardia. The test used in the determination of the output of the heart per beat was described by the authors in a previous article. It consists of simultaneous determinations of (a) the oxygen content of arterial and venous bloods; (b) the oxygen requirement of the tissues per minute; and (c) the pulse rate.

The authors made no actual observations on arterial blood since, in the absence of pulmonary congestion or heart lesions, it may rea-

sonably be assumed that the arterial blood is fairly completely saturated with oxygen.

The results show that in each case in this series, with one exception, the volume output of the heart beat was diminished. The average output was 55.3 c.c. per cent. The maximum and minimum values were 73 and 36.4, respectively. This series of cases has been compared with a group of diabetic patients showing acidosis. From a comparison of the two series the authors conclude that some factor other than increased metabolism must account for the excessive increase in pulse rate in cases of hyperthyroidism.

Bierring, Walter L.: Subacute Bacterial Endocarditis. Jour. Amer. Med. Assn., 1926, lxxxvii, 464.

The author describes the clinical findings of 30 cases of this disease observed during the last five years.

The author feels that a preexisting valve lesion is essential for the production of this form of endocarditis. The chronically involved valve seems to constitute a predisposing site on which the new infection is engrafted. In many instances the patients are not aware of the previous heart affection, as this is almost exclusively a disease of adult life. It is the valves of the left side of the heart that are chiefly involved. In the present series of 30 cases, the mitral valve was involved in 22, the aortic in 7, and in one case the lesion was of congenital nature. The previous disease, producing the original valvular affection, has, in more than one-half of the recorded cases, been an acute or subacute rheumatic infection. Twenty of the patients in this series gave such a previous history. Syphilis and scarlet fever and other infections are less frequent, while arteriosclerosis is rare as a specific lesion.

The disease seems to be incident to the period of adolescence and young adult life, occurring during the time of greatest incidence of valvular heart disease from all causes, excluding syphilis and arteriosclerosis. In the 30 cases there were 16 males and 14 females.

Between the production of the original valve lesion and the later reinfection, there is often an interval of several or many years of good health. To what extent the rheumatic history or the preexisting valve lesion itself produces a predisposition to subsequent infection or subacute bacterial endocarditis is a problem still unsolved.

Harrison, Tinsley Randolph, and Leonard, Bernard W.: The Effect of Digitalis on the Cardiac Output of Dogs and Its Bearing on the Action of the Drug in Heart Disease. Jour. Clin. Inves., 1926, iii, 1.

Experiments were carried out on dogs in a quiet state either after the administration of morphine or in dogs trained to experimental

conditions. Venous blood was drawn from the right ventricle and arterial blood from the left ventricle or from the femoral artery. The oxygen content of these samples was determined by means of the Van Slyke-Neil constant volume apparatus. Immediately after the blood samples were drawn the oxygen consumption was determined with the Benedict spirometer. The cardiac output per minute was calculated according to the Fick formula. Electrocardiograms were taken either immediately before or immediately after the determination of oxygen consumption. After controlled determinations of cardiac output the animals were given digifolin intramuscularly. The amount of the drug used varied in different experiments according to the weight of the animal and according to whether the therapeutic or toxic effect was desired. Subsequent examinations of the animals were made. Two experiments were carried out on trained animals with an increased metabolic rate which had been previously produced by feeding thyroid gland. One experiment was performed on a dog with elevated metabolic rate after frequent bleeding. These three animals had an increased cardiac output at the start of the experiment and offered an opportunity to study the effect of digitalis on the hyperdynamic heart. In two experiments in narcotized animals the usual procedure was carried out and subsequently the vagi were cut and the cardiac output again measured. In 4 experiments on trained unnarcotized animals, atropine was given after digitalization and the effect on the total output determined. In one experiment the rate of recovery from digitalization was studied by measuring the cardiac output every twenty-four to forty-eight hours until an approach to the normal level was found. In all, 19 experiments were carried out and 84 determinations of cardiac output were made.

Results.—Morphine caused a diminution of 8 per cent of the original cardiac output over a period of twenty-four hours. This is within the possible limits of error of the method, although the average variation in checked experiments is considerably less than this.

Effect on Pulse Rate.—No constant changes from therapeutic doses of digitalis were noted in morphinized dogs. In the 7 experiments on trained dogs the pulse rate was diminished in four instances and was increased in the other three. Toxic doses caused a marked increase in pulse rate in every instance.

Effect on Oxygen Consumption.—This remained fairly constant in most instances. In the morphinized dogs, a slight increase was noted several times as the experiment progressed. The trained dogs usually showed a decrease in oxygen consumption after digitalization. The oxygen consumption increased in 2 and decreased in 5, whereas the cardiac output decreased in every instance.

The Effect on the Arterial and Venous Oxygen Content.—Variations of 1 and 2 volumes per cent in arterial oxygen content were frequently encountered and in some of the longer experiments a drop of as much as 3 or 4 volumes per cent was found. In several instances the arterial oxygen content increased, then decreased and again increased. The venous oxygen content diminished after digitalization and the drop was greater than the arterial decline. The coefficient of utilization decreased in 3 instances, remained unchanged in 9 instances and increased in 45 instances. In a total of 57 it diminished after digitalis.

The Effect on the Cardiac Output.—In a total of 57 determinations after digitalis the minute output of the heart was increased in 3 instances and decreased in 54 instances. The increases were 3, 6 and 32 per cent. The first two figures are within the limits of error of the method. The increase of 32 per cent was obtained in one determination during an experiment in which the determinations yielded the usual decreased values for cardiac output.

The degree of decreased cardiac output was directly related to the amount of digitalis administered. The larger the dose of the drug, the greater the decrease in cardiac output.

The effect of digitalis on dogs with increased cardiac output was noted in 3 animals. The average change in output was 14 per cent, somewhat less than the average figure for normal dogs receiving similar doses of the drugs. The effect of digitalis in auricular fibrillation was studied in one animal who developed this disturbance after manipulation of the vagi. The experiment suggests that digitalis affects the output of the fibrillating heart in the same manner as the output of the normal heart but possibly in greater degree. From a study of these animals the authors believe that digitalis effects the constriction of the heart by direct myocardial action, but they do not regard this point as definitely established.